

Journal

OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

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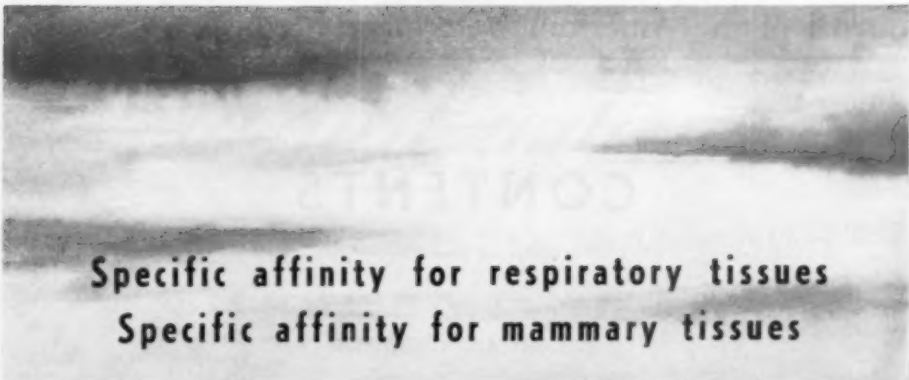


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Journal of the American Veterinary Medical Association

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Correspondence

July 29, 1958

Dear Sir:

Being specifically interested in the application of laboratory determinations to clinical medicine, I was pleased to read in the JOURNAL (July 15, 1958), two articles pertaining to erythrocyte determination in the Thoroughbred. However, comparison of the two articles accentuates the errors in the paper by Dr. Brenon. I would like to indicate just a few of these.

The erythrocyte count is always indicated in millions/cmm. (both papers incorrectly use millions/cc.). The packed cell volume (PCV) is expressed in per cent, not in mm., the expression Brenon uses, since some hematocrit tubes are not based on a 10-cm. calibration. The reference for this is any standard textbook of clinical pathology.

Many workers in laboratory medicine have discarded the erythrocyte count in favor of the far more accurate hemoglobin and hematocrit determinations. The erythrocyte count is grossly inaccurate, a fact which Dr. Irvine indicates. Our laboratory uses the erythrocyte count only in cases of anemia to determine corpuscular constants. This is an aid to the specific diagnosis of the anemia. Still, one wonders why Dr. Brenon indicates a lower erythrocyte count for a specific hemoglobin or hematocrit determination. In other words, he indicates a higher mean corpuscular hemoglobin (MCH) value and mean corpuscular volume (MCV) than do other workers. His figures may be computed to a MCH of 20.3 $\mu\text{g.}$ and a MCV of 62 cu. Values given by (or calculated from) the figures of Hansen, MacLeod, and Ponder; and Trum, Irvine, and Coffin are between 10.7 and 17.4 $\mu\text{g.}$ for the MCH and 37 to 53 cu for the MCV.

In table 1 of Dr. Brenon's paper, horse No. 3 has hemoglobin values of 15.2 and 20.5 Gm. before and after racing. However, the same blood samples have identical PCV values (70.0 and 70.0) and only a 1 per cent increase in the erythrocyte count (11.05 to 11.18 million). How does the author explain the increased hemoglobin in the absence of increased cell count and cell volume?

When a worker has obvious errors in his data, it is difficult for us to be convinced of the value of his conclusions.

Finally, I think Dr. Irvine is to be commended for a valuable study well done.

Very truly yours,
s/WALTER F. LOEB,
Ohio State University,
Columbus, Ohio.

[The unit of measurement for erythrocytes should have been million/cmm. See page 270 of this issue.—ED.]

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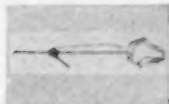
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equipment news!

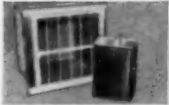
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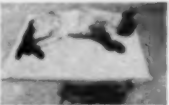
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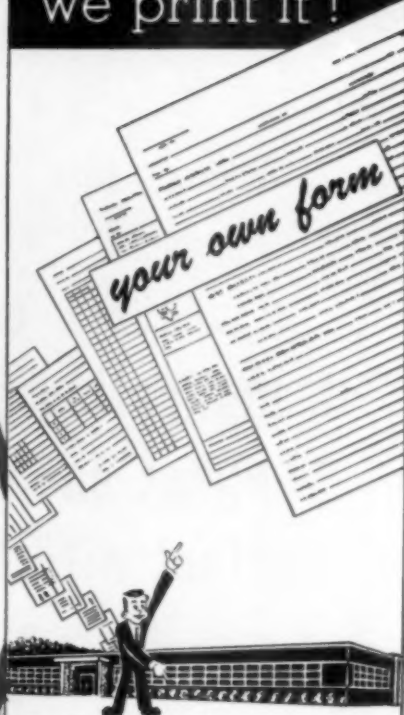
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Dr. Samuel F. Scheidy, veterinary medical director of Smith, Kline & French, Philadelphia, was elected to the office of president-elect at the Ninety-Fifth Annual Meeting of the AVMA in Philadelphia, and installed on August 20, 1958.



Dr. Samuel F. Scheidy

Graduating in 1929 from the School of Veterinary Medicine, University of Pennsylvania, Dr. Scheidy interned at the Veterinary Hospital of his alma mater for two years (1929-1931).

Following his internship, he was field veterinarian for the Hershey Creamery Co., in Harrisburg, Pa. (1931-1934) and for Abbott Dairies, Inc., Philadelphia (1934-1937). He was research associate (bovine mastitis) at the University of Pennsylvania from 1937 to 1938, and chief resident veterinarian and instructor in veterinary medicine at the University from 1938 to 1943.

In 1943, Dr. Scheidy became veterinary medical director of Sharp & Dohme, Philadelphia, and served in that capacity until July, 1957, when he joined the staff of Smith, Kline & French. From 1943 to 1955, he served part-time on the staff of the University of Pennsylvania as associate in medicine and assistant professor in clinical veterinary medicine.

Besides being a member of the AVMA, he holds membership in the Pennsylvania State V.M.A., Keystone V.M.A., United States Livestock Sanitary Association, the American Society of Animal Production, and Alpha Psi and Phi Zeta veterinary fraternities. He is also consultant to Medical Service at Large, Walter Reed Army Institute of Research, Washington, D. C.

Dr. and Mrs. Scheidy have resided in suburban Philadelphia for many years.

National Magazines Which Covered the Philadelphia Convention

Members of the national press attending the AVMA convention included:

Steven Spencer, Medical Editor, *Saturday Evening Post*

John Bird, Agriculture Editor, *Saturday Evening Post*

Gene Moore, *Time, Inc.*

Rodney Hohl, *Wall Street Journal*

Edwin Diamond, *Newsweek*

Phil McMullen, *United Press International Newsphotos*

Bill Anchaty, *Associated Press Newsphotos*

Edward McFall, *United Press International*

Noah Halper, *Associated Press*

John Rohlf, Livestock Editor, *Farm Journal*

Vern Schneider, Livestock Editor, *Successful Farming*


M. C. Gilpin, Editor, *Pennsylvania Farmer*

Gordon Berg, County Agent & Vo-Ag Teacher

Clarence W. Funk, *Farm Bureau Mirror*

John Robinson, Medical Writer, *Science Service*

Judy Randall, free lance writer, *Redbook Magazine*



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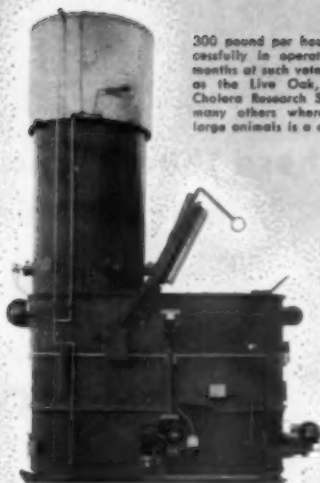
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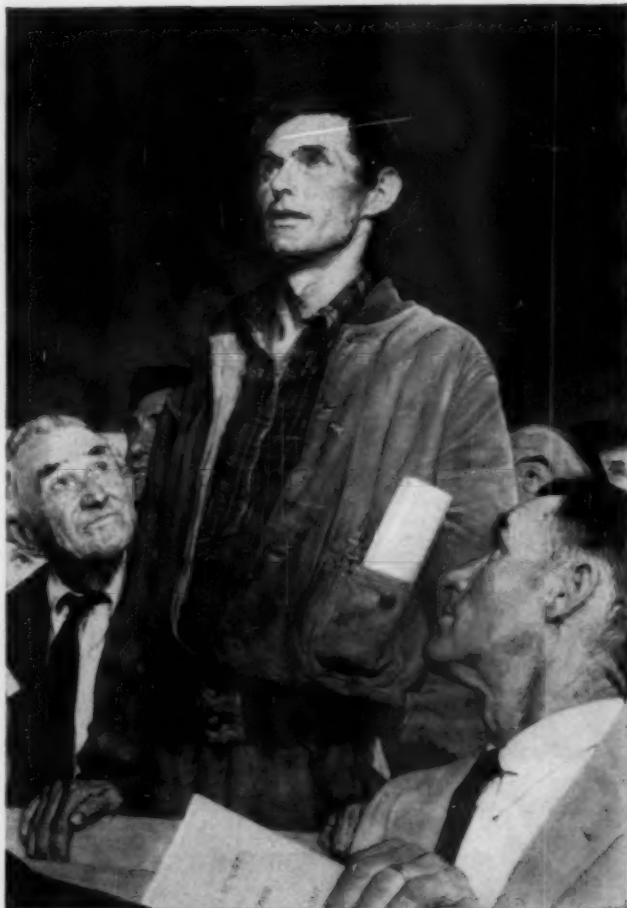
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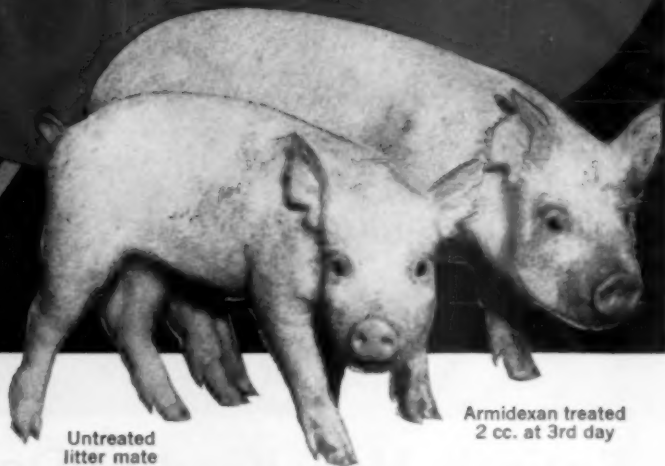
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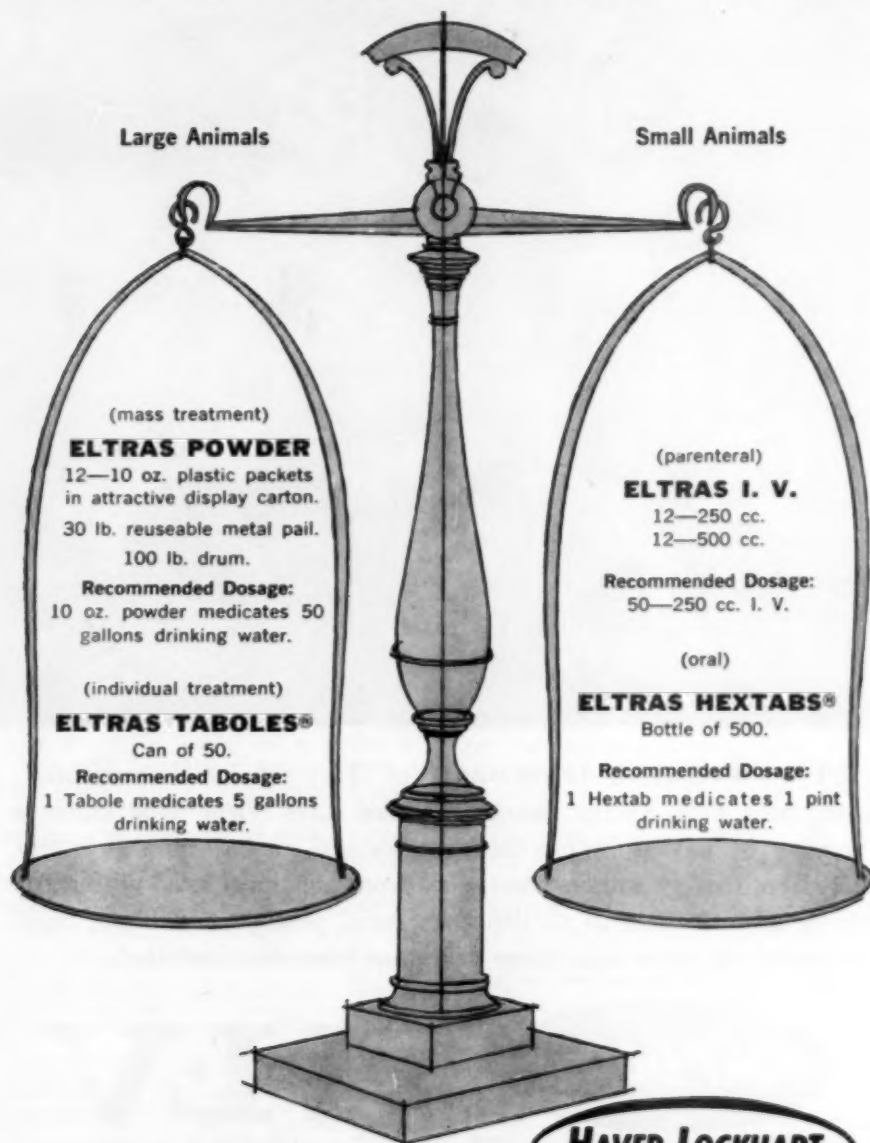


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Sertoli Cell Neoplasms in the Dog The Clinicopathological and Endocrinological Findings in Thirty-Seven Dogs

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Philadelphia, Pennsylvania

THE OCCURRENCE of Sertoli cell tumors in the testes of dogs has stimulated much interest ever since it was shown that animals affected with these tumors often develop a striking degree of feminization. There is general agreement that this is related to the presence of an excessive amount of estrogen, and there is strong evidence that the Sertoli cell is the site of estrogen production.

Several discussions of the clinical and histopathological features of Sertoli cell tumors in dogs have appeared in the literature.^{4-6,10} The tumors have also been reported in cats¹¹ and in a cockerel.¹⁷ Some of the earlier literature on carcinoma of the testes in dogs has been reviewed and 6 cases, in which the clinical syndromes resembled those seen with Sertoli cell tumors, were described.¹²

This report is based on a study of 37 dogs with this type of tumor, observed at the University of Pennsylvania Veterinary Hospital from July, 1952, to April, 1958. Since the preparation of this report, 3 more dogs with Sertoli cell tumors have been observed.¹⁸ Results of laboratory studies are presented which demonstrate that large amounts of estrogen are secreted by the tumors, and the relationship of

clinical signs to endocrinological alterations is discussed.

CLINICAL FINDINGS

Age and Breed Incidence.—The age (table 1) ranged from 3 to 20 years (av. 9.3), with 21 dogs between 6 and 12 years and only 6 under 6 years. This agrees with previously recorded observations.⁵

Skin Changes.—The initial complaint presented by the owner of a dog with a Sertoli cell tumor is often related to alopecia or other skin changes. This is particularly true when the neoplastic testicle is located in an abdominal position.

In 20 of the 37 cases reported here, there was alopecia. This tended to be symmetrical and involved primarily the ventral thorax and abdomen, the posterior and lateral aspects of the thighs and, occasionally, the neck and shoulder area if the animal wore a collar or harness. The hair was rather dry and brittle and could be easily pulled out. The skin was usually thin and velvety in texture.

In 3 dogs showing feminization, there was a diffuse, dusky purple pigmentation of the skin. Several other feminized dogs had a generalized pruritus. In 2 dogs, the skin was thick and tough and, on histological section, showed a great increase in collagenous fibers, characteristic of scleroderma. A generalized dermatomycosis was observed in 1 dog with marked feminization. The lesions, due to *Trichophyton mentagrophytes*, had been present intermittently for over one year.

Gynecomastia.—A dog was considered to be showing feminization if it had enlargement of the nipples, with or without thick-

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*See addendum on page 256.



Fig. 1.—Ventral view of the abdomen of a dog with a feminizing Sertoli cell tumor of an abdominal testis. Notice symmetrical alopecia and marked gynecomastia. The prepuce is edematous and flaccid.

ening of mammary tissue (fig. 1). Gynecomastia was observed in 16 animals, and the glands of several secreted a serous or milky fluid. In another dog, evidence of mammary secretion was observed histologically al-

though gynecomastia was not noticed clinically. Except in 1 case (case 12), all dogs with gynecomastia showed alopecia.

Attraction of Male Dogs.—Five dogs with feminization had a definite history of

TABLE 1—Some Characteristics of Dogs with Sertoli Cell Tumors

Case No.	Breed	Age (Yr.)	Testis affected*	Location of affected testis**
1	Wire-Haired Fox Terrier	9	R	S
2	French Poodle	8	?	A
3	Cocker Spaniel	6	R	I
4	Shetland Collie	9	L	A
5	Collie	8	R	S
6	Mongrel	8	Both	S,S
7	Boston Terrier	17	R	A
8	Collie	3	R	A
9	Boxer	6	R	I
10	Wire-Haired Fox Terrier	9	?	S
11	Mongrel	12	R	S
12	Boxer	6	L	A
13	Pomeranian	7	R	I
14	Boxer	9	Both	A,S
15	English Setter	13	R	S
16	Gordon Setter	12	Both	S
17	Pekingese	14	R	S
18	Boston Terrier	10	R	I
19	Boxer	6	R	A
20	Springer Spaniel	8	R	A
21	Mongrel	10	R	I
22	English Setter	9	R	S
23	Cocker Spaniel	10	Both	S,S
24	Collie	8	L	A
25	Mongrel	14	R	I
26	Welsh Terrier	6	?	A
27	Mongrel	14	?	S
28	Airedale	8	?	S
29	Poodle	7	R	A
30	Boxer	9	R	A
31	Mongrel	16	Both	S,S
32	Mongrel	10	L	A
33	Mixed	20	R	S
34	Mongrel	11	R	A
35	Boxer	9	?	S
36	Pomeranian	8	L	I
37	West Highland Terrier	13	R	S

*L = left, R = right; **S = scrotal, I = inguinal, A = abdominal.

attracting other male dogs. This may have been present to some degree in other instances without the owner being aware of it.

Prostate Gland.—Dogs that had Sertoli cell tumors but lacked feminization showed no significant changes in the prostate glands other than the usual hyperplastic and cystic changes often seen in the older dog. In dogs showing feminization, the prostate glands were atrophied in some and normal or enlarged in others. In 4 such dogs, marked prostatic enlargement, due to abscess formation, was an important part of the clinical syndrome (fig. 2).

One of these dogs (case 4) died of uremia and septicemia due to chronic urine retention and sepsis resulting from a large abscess of the prostate. In another dog (case 32), a large prostatic abscess had ruptured into the peritoneal cavity, causing fatal peritonitis.

Other Clinical Signs.—In many feminized dogs, the prepuces were flaccid, pendulous, and edematous; in others, they were somewhat atrophied. There were varying degrees of penile atrophy and, in some cases, loss of libido, marked lethargy and, according to their owners, premature aging. In 1 (case 4), there was a marked deposition of fat in the omentum and mesentery although the dog was cachectic and, as previously stated, died from uremia and septicemia.

NATURE OF SERTOLI CELL TUMORS

Testis Affected.—Of the 31 dogs on which information was available, the right testis

was involved in 22, the left in 5, and both in 4 (table 1).

Size of Affected Testis.—In most of these dogs, the Sertoli cell tumors were relatively large before the animal was presented for treatment. The largest neoplasm was in a Collie (case 24) and measured 12.5 by 12.5 by 12.5 cm.; the smallest, measuring 2.0 by 2.0 by 2.0 cm., was in a Boston Terrier (case 7). When the tumor was unilateral, the unaffected testicle was atrophied.

Unfortunately, accurate data on the ratio of tumor size to body weight was not obtained, so the relationship of tumor size to feminization was not determined. However, while feminization was usually observed only with tumors of a relatively large size, it was not present in some dogs with large neoplasms. The smallest tumor that produced feminization (case 12) measured 4.3 by 3.7 by 2.1 cm. This animal had gynecomastia but no alopecia.

Location of Affected Testis.—Of the 33 dogs with unilateral tumors, the affected gonads were located in an extrascrotal position (abdominal cavity or inguinal region) in 20 (table 1). In 3 of the 4 dogs with both testes affected, both were in the scrotum; in the fourth, one gonad was in the abdominal cavity. There appears to be a direct relationship between the presence of a Sertoli cell tumor in an extrascrotal testicle and the development of signs of feminization (table 2). In 81 per cent of dogs showing gynecomastia, the neoplastic testicle was in an extrascrotal position.

Biological Behavior of the Tumor.—In

Fig. 2—Abdominal organs of a Shetland Collie (case 4), showing a large abscess of the prostate gland (right center), the urinary bladder (top center), and the Sertoli cell tumor (lower center) which had caused feminization.





Fig. 3—Huge abdominal Sertoli cell carcinoma in an 8-year-old Collie. Notice the gynecomastia and the discrete metastatic lesions in the liver.

33 of the 37 affected dogs, the Sertoli cell tumors remained localized to the involved testicles although, in 6 of these, the tumors were classified histologically as carcinomas.

One of the 4 dogs with metastases was an 8-year-old, 50-lb. Collie (case 24) with a large Sertoli cell carcinoma of an abdominal testis associated with extensive metastases to the liver and spleen (fig. 3). Most of the parenchyma of the enlarged (10 lb.) liver was replaced by innumerable yellow tumor foci, some of which were umbilicated. In other instances, these foci had coalesced (fig. 4). The spleen weighed 1.75 lb. and contained many firm, tannish, raised nodules approximately 1 to 2 cm. in diameter.

TABLE 2—Relationship Between Location of Neoplastic Testicle and Signs of Feminization

Location of neoplastic testis	Dogs showing feminization		No. dogs showing no feminization
	(No.)	(%)	
Scrotal	3	20	12
Inguinal	4	57	3
Abdominal	9	69	4

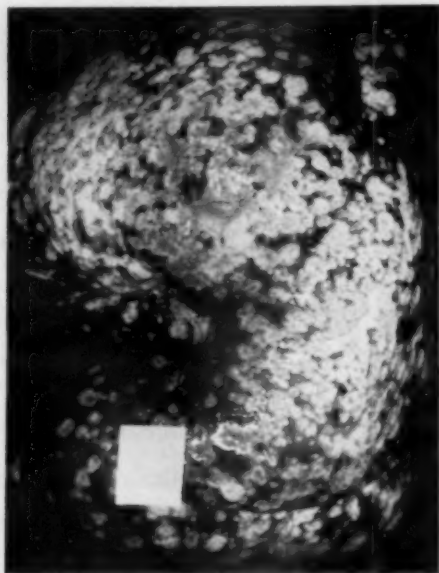


Fig. 4—Tremendous hepatomegaly due to widespread metastases from an abdominal Sertoli cell carcinoma (fig. 3). This liver constituted 20 per cent of the dog's weight.

A 45-lb., 12-year-old English Setter (case 22) showed clinical signs of metastasis after removal of the primary testicular tumor. This dog, when first seen, had a large tumor in a scrotal testis, with signs of feminization. Soon after the tumor, a Sertoli cell carcinoma (fig. 5), was surgically removed, the gynecomastia, sluggish behavior, and attraction of male dogs disappeared, only to return in approximately two months. After another few weeks, the dog's condition deteriorated rapidly and it died.

At necropsy, the iliac lymph nodes were greatly enlarged and completely replaced by metastatic Sertoli cell carcinoma (fig. 6). The affected nodes measured 4.2 by 2.2 by 2.5 cm. and 3.0 by 4.0 by 1.5 cm. This dog also had four other primary neoplasms (see "Concomitant Neoplasms").

A third dog (case 33) showed metastases to the inguinal, internal iliac, and mesenteric lymph nodes and to the right kidney. The primary tumor (6.0 by 5.0 by 4.5 cm.) was in the right scrotal testis of this 20-year-old mongrel.

A fourth dog, a 14-year-old terrier (case 27), had a primary tumor of a scrotal

testis with extensive metastatic lesions in the liver and pancreas.

PATHOLOGICAL CHANGES IN TESTICLES

The histopathological changes in testicles affected by Sertoli cell tumors have been described by others^{2,30,32} and, therefore, will be mentioned only briefly here.

The external surface of the Sertoli cell tumor is frequently nodular. On section, it is typically a brownish yellow to white, firm or hard mass that is irregularly lobulated by dense fibrous bands of tissue. Cysts of various sizes containing a clear fluid are commonly found. The cut surface frequently has a greasy texture (fig. 5).

The tumors exhibit considerable variation in structural and cellular characteristics. Although they are often arbitrarily divided into adenomas and carcinomas, many borderline cases occur.

The commonest structure is a tubular arrangement. However, in some cases, the cells may form cords or diffuse masses so that no tubular structure is recognizable.

In well-differentiated tumors, the cells are large, often elongated, and frequently possess cytoplasmic prolongations. The cytoplasm is eosinophilic, vacuolated, and foamy. The nuclei stain deeply and are narrow and elongated.

In less differentiated tumors, the cells lose the typical Sertoli cell characteristics and tend to be spherical or polyhedral, with an eosinophilic cytoplasm containing large, round, or oval nuclei.

One striking characteristic of all Sertoli cell tumors is the presence of large amounts of lipids, sometimes in the form of large droplets and globules. The lipid content per gram of Sertoli cell tumor may be three times as great as that in normal testicles.⁹ Histochemically, the lipids give many of the reactions characterizing steroid-producing tissues.³³

When the Sertoli cell tumor is unilateral, the opposite testis is atrophied (fig. 5), especially when feminization occurs. The testicular tissue is flaccid, dark brown, and may be almost absent.

CONCOMITANT NEOPLASMS

In 7 of the 11 animals with one or more neoplasms in addition to the Sertoli cell tumor, the additional tumor was located in the testis already affected or in the opposite testis. The concomitant testicular tumor was a seminoma in 4 dogs and an interstitial cell tumor in the other 3 dogs.

Dog 9, a 6-year-old Boxer with a history of generalized convulsions over a three-month period, had shown feminization and was attracting male dogs. Following removal of a Sertoli cell adenoma from the right inguinal region, there was a remission of the neurological signs for about three weeks; then convulsions became more

severe and the dog died. At necropsy, an astrocytoma measuring 1.8 by 2.3 cm. was found in the occipital lobe of the brain.

Dog 14, a 9-year-old Boxer, died six weeks after first showing neurological signs, characterized by spells of dizziness and ataxia. During these attacks, the dog salivated profusely, would fall upon its left side or back, and often urinated. It had a ravenous appetite and at times seemed to be blind.

At necropsy, the typical changes of feminization were observed in association with a large abdominal Sertoli cell tumor and a smaller Sertoli cell tumor of the scrotal testis. The pancreas contained two islet cell carcinomas—a grayish encapsulated mass (2.0 by 5.0 by 2.0 cm.) in the head of the gland and an oval tumor (1.3 by 1.2 by 1.0 cm.) in the body of the gland. The brain showed gross and microscopic evidence of a severe, diffuse encephalopathy.

Although blood sugar values were not obtained, it seems reasonable to suspect that the pancreatic islet cell adenocarcinomas (malignant insulinomas) were functional and led to hyperinsulinism, characterized by hypoglycemia.

Dog 18,³ a 10-year-old Boston Terrier, had three histologically different tumors of the external genitalia—a mastocytoma of the scrotum, and a seminoma and a Sertoli cell tumor of an inguinal testis.

Dog 22, a 9-year-old English Setter, had a malignant melanoma of the soft palate which had metastasized to the mandibular lymph nodes, parietal pleura, lungs, pericardium, both kidneys, the serosa of the ileum, and the pituitary gland. The cardia of the stomach contained a leiomyoma, and multiple papillary adenomas of bile duct origin were found in a small liver lobe surrounding the gallbladder. A huge cavernous hemangioma (21 by 17 by 9 cm.) was also present in the body of the spleen.

HORMONE ASSAY

In 6 dogs with feminizing Sertoli cell tumors, bioassays for total estrogen content in the urine were performed by the vaginal smear method.¹ In all instances, the preoperative urine samples contained between 44 and 64 mouse units (m.u.) of total estrogen for 24 hours. In 5 of these dogs, samples taken on the third or fourth postoperative day contained 12 m.u., or less, per 24 hours.

SURGICAL TREATMENT

Sertoli cell tumors were removed in 15 dogs showing feminization. Reports on 12 of the 15 indicated a remission of clinical signs in all 12. Gynecomastia receded, the coat reverted to normal within two or three months, and the diffuse dusky pigmentation of the skin disappeared. Dog 20 typically had showed marked gynecomastia and alopecia preoperatively; yet, three months after surgery, the nipples and coat had returned to normal.

In dog 22, the return of signs of feminization, after their regression following surgical removal of the neoplastic scrotal testis, was probably due to the iliac node metastases found at necropsy.

Postoperatively, in a few of the feminized dogs, there was a rapid disappearance of lethargy and a return of the penile sheath to normal size and tonicity. In some dogs, striking changes occurred within 24 hours.

Of the 10 dogs with nonfeminizing tu-

mors, the postsurgical results were good in 3, on which reports were available, and were probably good in all 10 since all tumors were classified as adenomas.

Prostatic abscesses were treated by direct aspiration of the prostate at the time of laparotomy, with subsequent injection of antibiotics into the abscess cavity. The pus was cultured and specific antibiotic therapy was used postoperatively.

DISCUSSION

There is evidence of at least three sites of estrogen production in the body: the ovary, the adrenal cortex, and the testicle. Since the urine of stallions contains large amounts of estrogen, while that of geldings contains little, the male gonads may normally secrete estrogen.¹⁰ Estrogen may also be extracted from the testes of bulls,⁷ and small quantities may be extracted from the testes of man.¹⁶

The cells responsible for estrogen production in the testes are not definitely known, but clinical and experimental data indicate that the hormone is produced by Sertoli cells. Dogs with Sertoli cell tumors show activity identical with that which can be produced by large doses of estrogen.¹²

Excessive amounts of estrogen are present in testicles containing Sertoli cell tumors and large amounts of the hormone appear in the urine of dogs affected with this neoplasm. Values of up to 70 μ g. of estrogen (expressed as *alpha*-estradiol) per kilogram of Sertoli cell tumor tissues have been reported in 2 dogs.¹⁰ The significance of this value can best be appreciated when compared to the 34 μ g. of estrogen (expressed as *alpha*-estradiol) which was extracted per kilogram of canine ovarian tissue at estrus.

There is little information on the amount of estrogen normally found in the urine of dogs, but the postoperative values obtained here are considered high. The assays were for total estrogen content of urine and do not reflect the type or types of estrogen which were present. No estrogenic activity was demonstrated in the urine of normal, anestrous bitches or male dogs.¹³ A value of 20 I.U. of estrogen per 24 hours was found in a dog with a feminizing syndrome in association with a Sertoli cell tumor.⁴ This tumor also contained a large amount of estrogen.

The marked fall in urine estrogen fol-



Fig. 5—Sertoli cell carcinoma (dog 22) of a scrotal testis, showing great enlargement of the neoplastic testis and many cystic areas. The opposite testis (above) shows advanced atrophy.

lowing removal of a Sertoli cell tumor indicates that the Sertoli cell is the site of estrogen production. It appears that in dog 22 the Sertoli cells in the metastatic lesion were functional and were the site of excessive estrogen production, since both testes had been previously removed.

More work, including estrogen assays for specific types of estrogens, is needed to establish quantitative relationships between hormone levels in Sertoli cell tumors and clinical signs. Some of the animals reported here showed a much more striking clinical picture than others. While critical data on estrogen levels is lacking, it seems reasonable to suppose that there is a direct relationship between (a) the hormone level and duration of estrogen action and (b) the intensity of such clinical signs as gynecomastia, alopecia, atrophy of the non-neoplastic testicle, and attraction of male dogs.

Other factors may be of importance in determining the clinical picture of dogs with functional Sertoli cell tumors. There may be quantitative differences between the sensitivity of tissues in different dogs in response to estrogen stimulation. Since the liver is an important site of estrogen breakdown, the presence of liver dysfunction in association with a functional Sertoli cell tumor might result in increased levels of circulating hormone. This may have occurred in dog 24.

Dogs injected with large doses of estrogen eventually develop an alopecia similar

to that seen in dogs with Sertoli cell tumors.¹² The primary effect of excessive estrogen on the skin is a retardation of hair growth, due to atrophy of hair follicles and sebaceous glands.^{6,15} The latter may account for the dry coat and pruritus observed in some animals. In dogs with Sertoli cell tumors, alopecia apparently develops as a result of failure of normal regeneration of hair, in association with loss of dead hair at sites of abrasion.

The gynecomastia observed in dogs with Sertoli cell tumors is undoubtedly the result of stimulation of nipples and mammary tissue by estrogens. The chief early effect of estrogen on the mammary gland is an extension and ramification of the ducts, with the occasional development of a few small lobules of alveolar tissue.

In a few dogs reported here, a small amount of milky fluid was observed at the nipple orifices, indicating that some lobular development had occurred. Others showed enlargement of the nipples without obvious gross involvement of mammary tissues. Spontaneous lactation was reported in 1 dog with a Sertoli cell tumor, and it was possible to induce lactation in 2 of 4 dogs by administration of prolactin.¹⁰

Large amounts of estrogen are known to inhibit the secretion of gonadotrophic hormones by the anterior pituitary gland, and gonadotropins are known to be essential for maintaining the size and functional

Fig. 6—Bilateral iliac lymph node metastases of a Sertoli cell carcinoma (dog 22). These metastatic lesions apparently caused a refeminization of the animal several months after castration. The urinary bladder is just below the paper marker and the two iliac nodes are to the left of the marker.



integrity of the normal testes. A reduction in gonadotropin secretion is presumed to be the factor mainly responsible for the testicular atrophy, characterized by degeneration of spermatogenic epithelium, in association with unilateral functional Sertoli cell tumors.

The size, and particularly the location, of the Sertoli cell tumor appear to be related to the development of feminization. The average size of the neoplastic gonads in dogs showing feminization was almost two to three times that of the affected testicles in dogs not exhibiting signs of feminization. However, the incidence of feminization was much higher when the Sertoli cell tumor was located in a retained testicle.

In some dogs, atrophy of thyroid glands was observed at necropsy. This, and also the sluggish behavior of some animals, may be due to the effect of excessive estrogen on the thyroid gland. Large amounts of estrogen are known to depress thyroid function,¹⁸ possibly due to inhibition of the thyrotropic hormone secreted by the anterior pituitary gland. The striking postoperative increase in activity in some animals may be related to release of this pituitary inhibition through a sudden fall in the circulating estrogen level.

There was a high incidence of right-sided neoplastic involvement in our series. We, and others,² have observed that right-sided cryptorchidism is much commoner than left. This may be related to its point of origin, since the right testis arises further cranially than the left and has a longer distance to traverse in order to reach the scrotum. Since cryptorchidism favors testicular neoplasia, there may be a direct relationship between the high incidence of right-sided neoplasia and cryptorchidism.

We have found that endocrine therapy has been uniformly unsuccessful in stimulating descent of the testes. Therefore, because of the definite correlation of cryptorchidism with neoplasia, as well as the genetic transfer of cryptorchidism, we advise bilateral castration of any affected dog.

Recently, tumor tissue obtained from 1 of these dogs (case 37) was grown in tissue culture, and successful transplants of the tumor to other dogs have been made.¹⁴ This work will be reported in detail at a later time.

ADDENDUM

Case 38.—An 8-year-old Boxer, which had developed a distinct feminization syndrome in six months and was being treated for nephritis, became so debilitated it was euthanatized. Necropsy revealed a large neoplastic right testis in the abdominal cavity. The histological diagnosis was Sertoli cell adenoma.

Case 39.—In a 6-year-old Boxer, a feminization syndrome developed in six months. The prepuce was enlarged. A large abdominal testicular tumor was removed surgically. The histological diagnosis was Sertoli cell tumor.

Case 40.—A 9-year-old Boxer with only the atrophic left testis in the scrotum had been vomiting for three weeks. A large abdominal mass was palpated. A large, irregularly-lobulated testicular tumor (20 by 12 by 10 cm.) was removed. There were no signs of metastases to any of the abdominal organs. Two months postoperatively, the dog was doing well. The tumor weighed 3.4 lb. and, on cut section, revealed large soft areas of hemorrhage and necrosis. The histological diagnosis was Sertoli cell carcinoma.

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Sertoli Cell Tumors and Alopecia in the Dog

Neoplasia of the Sertoli syncytium is not rare in male dogs 9 to 15 years old. When there is alopecia and feminization in dogs with displaced testicles, laparotomy and a search for the tumor is indicated.

Alopecia originating from involvement of the hypophysis, thyroid, or suprarenal gland are difficult to differentiate from each other but not from those originating from testicular tumors.

After surgical removal of the tumor, regrowth of hair usually starts in five to seven weeks unless there are metastatic complications.—R. Veilleux et al. in *Bull. Acad. vét. France* (Feb., 1958): 75.

Mammary Neoplasms of the Bitch.—Of the mammary tumors from 424 bitches, 249 were benign. Of these, 11 were of connective tissue type, 8 were simple adenomas, and the other 230 showed varying degrees of complexity. Of 187 malignant tumors, 87 were classified as carcinomas, 73 sarcomas, and 27 complex malignant tumors. Metastases were present in 41 of the 424 bitches.—*Vet. Bull.* (June, 1953): Item 1828.

Ringworm in Yugoslavia.—In a survey, ringworm was found in 0.4 per cent of 2,572 adult cattle, 6.0 per cent of 891 heifers, and 13.0 per cent of 843 calves. Of

the 165 cases, about 93 per cent were due to *Trichophyton verrucosum*, 7 per cent to *Trichophyton mentagrophytes*, and *Trichophyton violaceum* was found in 1 calf. In another area, 2.0 per cent of 2,559 cattle were infected. Ringworm was not found in 219 horses and in 995 sheep examined.—*Vet. Bull.* (May, 1958): Item 1392.

Rinderpest in Sheep in Nigeria

Rinderpest is often said to cause natural infection in sheep and goats but recent reports of such infection are rare. In July, 1957, the disease appeared in cattle in intimate contact with native sheep. Eight days later, 54 of the 110 sheep were sick and 12 died in two days from serologically confirmed rinderpest.

It was controlled in the cattle by vaccinating with lapinized rinderpest virus plus slaughter of the affected animals. However, this procedure failed to control the disease in the sheep so the entire flock was slaughtered. The course of the disease in sheep was about five days but some were affected only mildly and probably would have recovered.

Although rinderpest has been widespread among the cattle of the region for many years, and although they generally graze with sheep and goats, this is the first report of the disease in exposed sheep. This may have been due to: (1) the excellent condition of the sheep which were grazing on luxuriant pasture (lesions are more severe in animals on a high plane of nutrition); (2) to the close contact and intensive grazing on a limited area; or (3) to a heavy infection of Oesophagostomum worms, with damage to the intestinal mucosa.—R. H. Johnson in *Vet. Rec.* (May 31, 1958): 457.

Comment.—The hypothesis that stress factors lowered the innate resistance of the above sheep is interesting but not supported by evidence. A dose of virus just capable of infecting a susceptible host induces a disease indistinguishable from that induced by a vastly greater dose except that the incubation period is longer with the smaller dose. The infection in these sheep could be attributed to a more virulent strain of virus. The prompt slaughter of the flock would probably remove that strain from the epizootiological picture.—G. R. Scott in *Vet. Rec.* (June 21, 1958): 521.

Defects—Not Fractures—of the Fibulae in the Horse

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AN ARTICLE on fractures of the fibulae of horses was published in 1956.⁵ This report was based on 38 horses with conditions characterized by lameness after hard work or racing, return of lameness following a period of rest, an abnormal gait, breaking of gait, and usually myositis of the lumbar and gluteal muscle groups. The fracture was fairly constant in location, and delayed healing or nonunion was common.

This article aroused our interest, for the clinical characteristics described have generally been assigned to early spavin lameness. The frequency of the condition at this clinic was indicated in a series of 100 hospitalized horses during six months in 1956. This was a sampling of hospital cases of both lame and sound horses and, among the group, 68 per cent were found to have this condition. Again in the fall of 1957, the records of another group of 44 lame horses with fibular defects were assembled in four months. This last group consisted of 22 Standard-bred trotters, 15 Standard-bred pacers, 5 Thoroughbreds, 1 Saddle Palomino, and 1 American Saddlebred horse.

The 1956 report appeared to gain wide acceptance and the condition was considered a new entity by some. However, the medical artist has recorded these "fractures" in our anatomy textbooks. Reverchon's "Anatomie du Cheval, Osteologie et Myologie," published in 1849, clearly demonstrates in plate 2, figure 3, a fused fractured fibula bone. Another nineteenth century monograph by Brunot entitled "Etudes Anatomiques du Cheval" reveals in plates 4, 9, and 12 the presence of "fractured" fibulae that have fused.

These have been described^{1,3,6,7} by others. The older portrayals of this defect, and the high incidence of this uniform lesion, do not seem to fit into the classical textbook description of fractures.^{7,9}

In the nineteenth century, the fibula was thought to have developed from a single center of ossification. However, three cen-

ters of development are now accepted:⁸ the proximal extremity or head, the shaft, and the distal extremity, which early in fetal life becomes the lateral malleolus of the tibia.

The time of fusion of the head to the shaft is not given in modern textbooks of anatomy. However, it has been estimated that 45 per cent are fused by the eighth year, 51 per cent by the fifteenth year, and 78 per cent by the twenty-third year.⁹

In an effort to ascertain fusion time between the head and shaft of the fibula, a series of radiographs was taken on young animals in the clinic. These studies indicated that fusion may not be complete at 42 months of age in horses not doing work, heavy training, or racing. All fibulae studied in immature horses followed a similar pattern of development, i.e., simultaneous growth from both the head and shaft of the fibula. In tracing the development of the head and shaft, it was apparent that fusion would occur between 4 to 6 cm. from the proximal extremity in most cases.

In some lame horses, 12 to 18 years of age, the head and shaft had apparently failed to fuse, yet the horses had been lame for only a short time, with no history of chronic lameness. This appears to contradict the opinion that the condition was a fracture.

Anatomic dissections of the fibular region revealed that at least three muscles—the lateral extensor, the tibialis anterior, and the flexor hallucis (part of the deep digital flexor)—are attached directly or indirectly to the fibular head and shaft. When these muscles were reflected, the interosseous ligament was found to vary from existing anatomic descriptions. The major tibiofibular ligaments were located in sites which had a high correlation to the most common locations of the fibular defect (fig. 1).

Heavy stress exists at the points of the ligamentous attachment between the fibula and tibia, where slight organized periostitis, at ligamentous insertions on the tibia, was seen in some radiographs of older horses. The muscles surrounding the fibula

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would appear to pull the distal end of the head and shaft down and away from the lateral surface of the tibia.

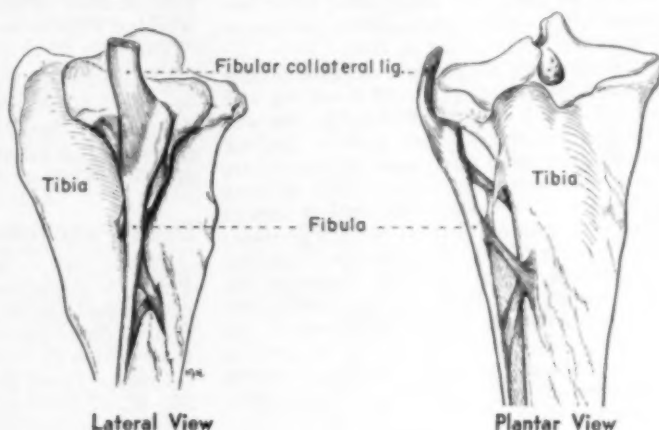
This motion is confirmed by radiographic measurements which reveal an oblique defect with a 4.24-mm. deviation from a transverse line in 104 fibulae. This figure

hyperemia, as indicated by an increased number of barium-injected vessels, was found.

Finally, microscopic studies revealed the presence of epiphyseal cartilaginous plates across the defects in the fibulae.

It is possible that muscular action and

Fig. 1.—Sketch showing the lateral and plantar views of the tibia and fibula of a horse. The head of the fibula is fixed in position by the lateral collateral ligament of the stifle joint and by three tibiofibular ligaments.



was arrived at by measuring from the fibular head to the lateral and medial levels of the defect. The fibular head is fixed in position by the lateral collateral ligament of the stifle joint, as well as three small strong tibiofibular ligaments (fig. 1).

In fresh specimens, little if any motion is visible, which agrees with Bradley² who states, "A strong short (joint capsule) binds the head of the fibula so closely to the lateral condyle of the tibia as to prevent anything but the smallest degree of movement. Not infrequently the capsule ossifies with age." Ligamentous attachments plus capsule ossification would indicate motion of the distal fragment rather than of the fibular head.

Further study of radiographs of the fibula does not support the fracture theory, for no callous formation in the form of new periosteal bone is found. Without bony changes visible on the radiograph, the next bit of proof would be the study of the arterial system for evidence of hyperemia.

The arterial system of a leg from a horse with an asymptomatic fibular fracture was injected with a barium sulfate suspension and then radiographed. No radiological evidence of hyperemia existed. The leg was then dissected and no visible evidence of

ligamentous attachment prevent normal bone development and subsequent ossification and fusion between the fibular head and shaft but, more probably, there are more centers of ossification than previously described.

SUMMARY

Evidence is presented which indicates that fibular defects are of a developmental, rather than traumatic, origin.

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Discontinuity of the Equine Fibula

A clinical and radiographic study of interruptions of the fibulae of horses was started in 1952 and accelerated after articles on the subject appeared in the American literature, starting in 1956.

Radiographs were taken of both fibulae in 120 horses from 1½ to 23 years old, and a total of 250 native cross-bred horses was studied. In over 50 per cent of healthy horses, the head and body of the fibula were not continuous at any time of their existence. The radiographs showed normal fibulae in both limbs in 51 (42.5%) horses; in the other 69, the disruption was present in 1 fibula in 20, in both fibulae in 49. There were 2 or more interruptions in the fibulae in 19 animals. In two thirds of the animals, the disruption was in the area of the proximal epiphyseal cartilage and in the others it varied as far as 12.5 cm. distal from the head.

In 4 horses in which the fibula was exposed and experimentally fractured, there was difficulty in extending the limb forward, particularly when trotting, and a slight dragging of the toe similar to that with paralysis of the peroneal nerve. Regardless of the amount of injury, lameness was always of the same type and of short duration. Because of the movability and flexibility of the distal portion of the fibula, an attempt to fracture it by an external blow was unsuccessful.

It is concluded that the discontinuity, which has been diagnosed as a fracture, represents a normal phenomenon which occurs in approximately 50 per cent of healthy horses.—B. Zeskov in *Brit. Vet. J.* (April, 1958): 145.

Chloral Hydrate for Equine Surgery

During the Burma campaign (World War II), it became necessary to silence the braying and whinnying of the animals to be used close to enemy lines. The vocal cords of a few old mules were experimentally excised through the thyroid notch. Since there was considerably less vigorous breathing and spraying of hemorrhage under chloral hydrate anesthesia than with ether anesthesia, the former was used.

Surgery was performed successfully on several thousand animals. In one group,

there was delayed jugular periphlebitis, probably due to the method of using a casting rope which caused pressure on the vein, causing a leakage of blood and chloral hydrate through the needle puncture. These, and a few cases where the drug was deposited outside of the vein, were successfully treated by incising the swelling and washing with saline solution.

The dose of chloral hydrate varied from 0.5 oz. in 8.0 oz. of water to 1.0 oz. in 16.0 oz. of water, depending on the size of the animal and the duration of anesthesia required.—C. M. Stewart in *Vet. Rec.* (May 17, 1958): 430.

Blood Transfusion Shock in Cattle

Twenty cattle, in which shock was produced by transfusion of incompatible citrated blood (up to 65 ml. per minute), in Russia, recovered and were slaughtered for examination 24 hours later.

Lesions found in those given more than 160 ml. of blood were subpleural petechiae, often accompanied by pulmonary edema and, in some cases, swelling of the kidneys and petechiae in the heart muscle and liver. Histologically, there were hemorrhages in the alveolar walls and interstitial tissue of the lungs, hyperemia in the kidneys, and degenerative changes in kidneys and liver.

The shock and lesions were less in 3 cattle given 1,100 to 1,600 ml. of incompatible blood stabilized with 100 ml. per liter of 15 per cent solution of calcium chloride instead of sodium citrate. There was neither shock nor histological changes in cattle transfused with compatible blood.—*Vet. Bull.* (May, 1958): Item 1468.

Effect of Temperature on Survival of Bacteria in Blood for Transfusion.—The problem of contamination of blood for transfusion was investigated, particularly the effect of temperature on contaminating organisms. Bacteria which were isolated from infected blood, and grew at 4 and 37 C., were inoculated into samples of freshly drawn blood in an attempt to reproduce the actual mode of infection during donation. It was demonstrated that, although experimentally there are slight advantages in refrigerating blood within 30 minutes of collection, in practice, refrigeration in temperate climates is not essential until at least eight hours after collection.—*Vet. Bull.* (June, 1958): Item 1727.

Removal of Magnetic Foreign Bodies by Paracentesis

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PERHAPS NOTHING has added more impetus to the practical and economic management of traumatic reticulitis, so-called "hardware disease," than the advent of the Alnico V[®] magnet.

This technological advance, coupled with the providential coincidence that the greatest percentage of metallic foreign bodies are of ferrous alloys, has made removal of the foreign body possible without use of the classic rumenotomy.

In an effort to overcome major surgical difficulties arising from rumenotomy, the author has modified the procedure to minor surgery, which might be termed "a micro-rumenotomy." At the same time, the technique has been kept simple and inexpensive.

An apparatus (fig. 1) of the following design† is used: Basically, the instrument consists of five component parts—(1) a complete trocar assembly; (2) an aluminum alloy tube which will pass through the trocar cannula; (3) a jointed aluminum alloy rod which is housed inside the tube (the rod is so designed that the operator can follow movements of the magnet within the reticulum); (4) a special magnet designed to give the greatest pull is attached to the end of the rod (the magnets can be replaced by any machinist); and (5) a plastic tube which acts as a carrying case.

TECHNIQUE

The animal is restrained with a nose lead, the head being drawn around to the right. In a row of stanchions, the cow to the left is removed to furnish ample room for the operator.

The paralumbar fossa is washed and disinfected. After the skin and muscle are infiltrated with a local anesthetic, a 3-inch vertical incision is made. Through this opening the rumenotomy trocar is thrust into the rumen and directed toward the right olecranon (fig. 2).

Dr. Carlson is a general practitioner in Acton, Mass. The author thanks Dr. Roy D. Hoffman, Bedford, Pa., for professional and technical advice in the development of this instrument.

†Produced by Allied Sales Associates, Long Beach, Calif.
†Instrument incorporating this design is manufactured by Bedford Laboratories, Bedford, Pa.



Fig. 1—Sketch showing the instrument, in situ, for removing metallic foreign bodies from the reticulum of a cow.



Fig. 2—Insertion of the trocar-cannula assembly into the reticulum of a cow.

The instrument is then inserted into the cannula about cannula length (fig. 3) and, by manipulating the butt end of the instrument, the angle of the cannula is brought into an almost horizontal plane, then passed along the dorsum of the rumen, over the anterior ventral pillar, and down into the reticulum, as ascertained by exerting gentle pressure forward on the instrument. Definite pulsation of the heart will be felt on the instrument if it is in proper position.

At this point, the inner rod is pushed forward to loosen the magnet from the end of the tube, allowing it to lie free on the floor of the reticulum (fig. 1). The instrument is left in this position for approximately a minute, allowing the contractions to move it about. Then the reticulum is

"swept" by appropriate movements of the butt end of the instrument.

The magnet is withdrawn into the end of the tube and the entire instrument slowly withdrawn through the cannula. Magnetic foreign bodies will cling to the magnet (fig. 4)—checking with a metal detector will tell whether all of the metal has been removed. Sometimes a number of insertions and withdrawals are necessary to remove all of the metal.

In addition to this method, the instrument is placed as first described, and the inside rod pushed forward as the outside tube is drawn back until the first joint in the rod is reached. Then the entire apparatus is pushed forward and slightly upward until the bend in the joint rests lightly against the anterior wall of the reticulum. In this position, by manipulating the joint

Fig. 3—Insertion of the instrument through the cannula into the reticulum of a cow.

Fig. 4—Magnetic foreign bodies clinging to the magnet after removal from the reticulum of a cow.



on the proximal end of the instrument sideways, the magnet is "swept" across the wall and floor of the reticulum. Also, if there is evidence of metal in the rumen, the instrument is directed ventrally into the rumen and all parts are "swept."

Subsequent to the operative procedure, medication might be administered through the cannula, depending upon the condition of the animal at the time of the operation, according to the judgment of the clinician. After removal of the cannula, the incision is packed with a suitable wound powder and the animal is given a parenteral antibiotic or chemotherapeutic agent as a prophylactic measure. No suturing is necessary. In fly season an appropriate repellent should be applied. Little or no aftercare is needed.

SUMMARY

A simple method and apparatus for removing magnetic foreign bodies from the rumen and reticulum of the bovine stomach are described.

Bovine Dystocia

Records kept on 2,097 calvings in several large dairy herds, in England, which were free of reproductive diseases, show that dystocia occurred in 95 (4.5%). Fifty dystocias were in the 555 Holstein-Friesian cows and heifers, an incidence of 9 per cent; and 45 were in the 1,542 Ayrshires (3%). Fetal membranes were retained for more than 48 hours after 96 parturitions (4.5%); if dystocia and twin pregnancy cases were excluded, the incidence would be 3.0 per cent.

Of 200 cesarotomies reviewed, maternal immaturity was responsible for 9 per cent, fetal oversize for 19 per cent, incomplete cervical dilatation for 21 per cent, fetal monsters for 22 per cent, uterine torsion for 15 per cent, hydrops of the fetus or its membranes for 5 per cent, postural obstruction for 5 per cent, transverse presentation for 1 per cent, resorbed mummies for 2 per cent, and pelvic injury was responsible for 1 per cent. When the fetus was alive, the membranes were already or easily detached in 1.6 per cent, were firmly attached but expelled naturally in 70.0 per cent, and were retained for more than 24 hours in 28.0 per cent. When the fetus was dead, the membranes were already or easily detached in 60 per cent, firmly attached

but expelled naturally in 15 per cent, and retained for 24 hours or more in 25 per cent.

Records from all sources indicate that fetal oversize is of greater significance in the Holstein-Friesian breed than in other dairy breeds. Data compiled both in England and in the United States give an average fetal weight of about 90 lb. in this breed, 71 lb. in Ayrshires, 76 lb. in dairy Shorthorns, 71 lb. in Guernseys, and 55 lb. in Jerseys, with male calves about 5 per cent heavier than females.

The cause of failure of the cervix to dilate fully is unknown but would seem to be due to hormonal dysfunction. It is often ascribed to fibrosis due to injury at previous parturitions but it occurs as frequently in primigravida as in multigravida; furthermore, the latter may subsequently calve normally. Since straining is usually insignificant, the fetus may have been dead for some time and the prognosis grave before veterinary attention was requested.

Uterine torsions occurred to either side but more often to the left. Torsion varied from 90 to 180 degrees, the latter being more common in multipara. The incidence did not vary significantly with the breed or when the animals were pastured or stabled. Torsion seemed more frequent when the calf was exceptionally heavy. It probably occurs in the first stage of labor, during dilation of the cervix, and when the calf's movements are particularly vigorous. It seems doubtful that the cervix could dilate after torsion has occurred; in 90-degree torsion, the limbs are frequently found extending through the cervix. The greater frequency of 180-degree torsion in multipara may be due to the greater volume of the abdomen. Since labor was usually absent or mild, the calves were dead (often putrid) in 75 per cent of the referred cases.

Following cesarotomy, 80 to 90 per cent of the cows made an apparent recovery but, if convalescence was stormy, lactation was often unsatisfactory. Of the calves which were alive and normal when removed, 80 per cent survived.—J. G. Wright in *Vet. Rec.* (April 26, 1958): 347.

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Correction of Uterine Torsion (a comment stimulated by the above article).—Having determined the direction of rotation, the well-lubricated hand (left hand if the torsion is clockwise, right hand if

anticlockwise) is inserted, following the twist until the hand enters the uterus. The operator is now bending forward with the palm of the hand upward. The torsion may then be corrected by grasping any available part of the fetus and twisting it while assuming the normal upright posture. Correction will be facilitated if a swinging or rocking movement of the uterus can be started. This method is preferred to rolling the cow, since there is no risk of increasing the torsion, and it requires no equipment or lay assistance.—*T. S. Lloyd in Vet. Rec. (June 28, 1958): 544.*

Cesareotomy in the Cow.—During three years, in Norway, 68 cesareotomies were performed on cows and heifers, with complete recovery in 75 per cent. In cases of torsion of the uterus (26%) and in prolonged delivery with emphysematous fetuses (19%), the recovery rate was 61 per cent.

Five cows with hydramnios or hydrallantois were operated on after gradual aspiration of the fluid, during a 30- to 60-minute period, with recovery of 4 cows and delivery of 1 live calf. All cows operated on for other reasons recovered.

For cesareotomy, the cow was anesthetized with 25 cc. of a 2 per cent xylocaine solution, given epidurally, and supplemented by local infiltration. It was then cast on its right side and an incision was made longitudinally, 8 to 20 cm. lateral to the left "milk vein." The fetal membranes were retained in 60 per cent.—*N. O. Rasbech in Nord. Vet.-med. (Oct., 1957): 721.*

Fetal Dystocia Due to a Hereditary Defect.—Fetal dystocia due to a greatly distended abdomen was observed in 6 Swedish Highland cattle. In each, an atresia of the ileum had caused a dilatation of the intestine to an average diameter of 5 to 6 cm., due to an accumulation of contents. The lethal defect was accompanied by premature parturition (at 7 to 8 months). The calves were all inbred descendants of 1 bull.—*B. Nihleen and K. Ericksson in Nord. Vet.-med. (March, 1958): 113.*

Artificial Insemination in Swine

Artificial insemination is not as practical in swine as it is in cattle. Swine breeding is seasonal, although some breeders breed sows every month. The cost of purchasing

and maintaining boars for natural breeding is relatively much less than for bulls. In Iowa alone, 75,000 to 90,000 boars are sold annually, but perhaps 15 per cent are returned as unsatisfactory breeders, largely due to mismanagement. Boars frequently spread disease since many are purchased without knowledge of the health of the herd from which they come.

A bull ejaculates 5 to 10 ml. of semen containing 800 million to $1\frac{1}{2}$ billion sperm per milliliter. A boar ejaculates 200 to 500 ml. of semen containing about 100,000 sperm per milliliter. Semen from a bull is uniform whereas one ejaculum from a boar will contain some watery semen and other portions which may resemble "cooked tapioca."

For use, boar semen must be filtered; a satisfactory diluent has not been developed. A former artificial inseminator of cattle, in Wisconsin, is getting satisfactory results using insemination on a few herds of swine. With the changing methods of management in swine production, artificial insemination could become practical.—*J. B. Herrick in A. I. Digest (May, 1958): 19.*

Control of Vaginal Prolapse in Ewes

The incidence of vaginal prolapse in ewes, in Great Britain, is given as 0.53 per cent but may be 20.0 per cent in some flocks. A hereditary factor may be involved, possibly as an endocrine imbalance or an anatomical peculiarity.

In one flock, with a history of up to 4.8 per cent of the ewes being affected in previous years, a new method of treatment was tried. In 18 cases, after the prolapse was reduced, a stainless steel wire device was inserted to prevent recurrence. It is shaped as an elongated letter "U" with the arms bent laterally at right angles and with eyelets at each end for tying to a harness. Results were completely satisfactory in 16 ewes but 2 had to be slaughtered.

No prolapses occurred in a second flock of different breeding kept separately on the same farm and under the same management. This may indicate the effect of a hereditary factor although, due to a slight difference in feeding, there was more bloating in the affected flock.—*B. V. Jones in Vet. Rec. (April 26, 1958): 362.*

Report of a Field Trial on the Use of Phenothiazine Preparations in Feedlot Cattle

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IN RECENT MONTHS, there has been increased emphasis placed on the treatment of feedlot cattle for internal parasites. The drug most widely used for this purpose is phenothiazine. Since individual treatment of animals offers certain problems in restraint and handling, there has been a demand for a suitable product which could be incorporated in feed.

Phenothiazine, however, is quite unpalatable to livestock unless it is present in low concentration. Because of this, there have been attempts to obviate this feature of phenothiazine by mixing it with molasses, adding flavoring ingredients such as anise, or combining it with such compounds as carob flour.

In addition to the claims of palatability for such mixtures, claims have been made concerning an enhancement of the anthelmintic efficiency and a reduction in toxicity of phenothiazine when carob flour is used as an additive.⁵ The basis for these claims, and consequently the claims themselves, have been questioned.^{1,6}

The present trial had three principal objectives: (1) to evaluate the palatability of two rather widely used products, one a flavored phenothiazine liquid preparation,* the other a carob flour-phenothiazine mixture;⁶ (2) to obtain some evaluation of the efficiency and toxicity of the products when used in feedlot cattle; and (3) to determine if treatment of cattle such as those used in the present trial was economically sound.

MATERIALS AND METHODS

The 194 yearling steers used were located at a feedlot near Rio Vista, Calif. They had been brought to the feedlot from Elko, Nev., on Oct. 29, 1956, and were fed pea silage and a mixture of alfalfa and volunteer hay. After 80 days, they

were placed on a fattening ration and brought to full feed.

On November 28, they were allotted at random to four groups and each group was weighed. In addition, fecal samples were taken from 10 animals in each group for quantitative parasite egg counts.

The treatment of the animals, beginning on December 4, was as follows:

Pen 11.—The carob-flour compound (3 lb. per day for three days) was spread over the pea silage (approximately 15 lb. per animal). This was equivalent to a total dosage of 60 Gm. of phenothiazine per head.

Pen 12.—Three gallons of the flavored phenothiazine liquid preparation was spread over the surface of the pea silage and mixed thoroughly with a pitch fork. This was equivalent to a single dose of 60 Gm. of phenothiazine per head.

Pen 13.—The animals in this group were left untreated to serve as controls.

Pen 14.—These steers were each drenched with 60 Gm. of phenothiazine.**

The animals in each group were weighed five times after treatment, the final weight being taken on the day the "finished" animals in each group were "cut" out for slaughter.

At the time of slaughter, the abomasum and small intestines of 10 animals from each group were obtained and nematode counts made by methods previously described for sheep.⁷

RESULTS

Because of the low parasite egg counts at the start of the trial (table 1) and the limited value of egg counts in the evaluation of phenothiazine as an anthelmintic, no further fecal examinations were made. There were no significant differences between the mean egg counts of the four groups.

The following observations were made at the time of treatment by the herdsman and by us.

Pen 11.—The animals ate only two thirds as much feed on the first two days after treatment and did not return to full feed until the fifth day.

On the second day of treatment, 40 to 60 per cent of the steers were showing variable degrees of pink discoloration of the preputial hair. Only 2 or 3 showed coloring

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*The liquid preparation was supplied through the courtesy of Atomic Basic Chemical, Inc., Pittsburgh, Pa., and the carob flour-phenothiazine through the courtesy of Vetrochem Co., Berkeley, Calif.

**Product of H. C. Burns Co., Inc., Oakland, Calif.

TABLE 1—The Numbers, Weights, and Parasite Ova Counts of Steers at the Start of the Trial

Pen No.	Treatment	Total phenothiazine (Gm.)	No. of animals	Av. weight (lb.)	Av. fecal parasite egg count (eggs/Gm.)
11	Carob flour preparation for 3 days	60	48	699	19
12	Flavored liquid preparation	60	49	696	40
13	None	—	47	688	22
14	Drench material	60	50	690	11

as intense as that uniformly shown by the steers in pen 14.

On the third day of treatment, 60 to 70 per cent of the animals showed variable degrees of hair discoloration, with an increased number showing discoloration as intense as those in pen 14, which showed greater discoloration than on the previous day.

On the fourth day, all of the animals showed hair discoloration, some of them more, some less, intense than any of the animals in pen 14, the variability being quite noticeable.

Pen 12.—The steers in this group ate only one half as much as they normally would have consumed during the first 24 hours following treatment. As a result, the herdsman added feed and, consequently,

70 to 80 per cent of the animals were showing discoloration, and the discoloration was more intense than in the animals in pen 11. By the third day, no discernible difference between those in pen 11 and pen 12 could be made with regard to hair discoloration.

Pen 13.—At no time did this group fail to eat the normal amount of feed, nor did they show any discoloration of preputial hair.

Pen 14.—The animals in this group ate the normal amount of feed at all times. Preputial hair discoloration was uniform among all steers and reached maximum intensity by the third or fourth day after treatment. Feces of treated animals in all groups became darker after exposure to air than feces of control animals, but no red color was discernible.

TABLE 2—Average Weight of the Steers at Different Weighings

Pen	Weight (lb.) of steers on:						
	Nov. 28	Dec. 19	Jan. 10	Feb. 8	Mar. 22	June 11	June 24
11	699	720	761	801	930	1,104	—
12	696	724	751	802	925	1,103*	1,111
13	688	721	746	775	906	1,090*	1,109
14	690	715	742	785	923	1,095*	1,122

*Obtained by extrapolation.

the medicated feed was not totally consumed until the second night. As in pen 11, they did not return to full feed until the fifth day.

Observations on the discoloration of preputial hair were similar to those in pen 11 with the exception that, on the second day,

The data concerning average weights and weight gains in the four groups is presented (table 2, 3). At the final weighing, steers considered ready for market were "cut" from each group as follows: 32 from pen 11; 35 from pen 12; 37 from pen 13; and 36 from pen 14. An experi-

TABLE 3—Weight Gains Between Weighings, Total Weight Gain, and Daily Gain for the Entire Period

Pen	Weight gain (lb.) between weighings					Total gain to June 11	Total gain at slaughter	Net daily gain at slaughter
	Nov. 28- Dec. 19	Dec. 19- Jan. 10	Jan. 10- Feb. 8	Feb. 8- Mar. 22	Mar. 22- June 11			
11	21	41	40	129	174	405	405	2.07
12	28	37	91	123	178*	407	415	2.09
13	33	25	29	131	186*	402	421	2.05
14	23	29	43	138	172*	405	432	2.07

*Obtained by extrapolation.

enced cattle buyer "cut" the cattle in all groups and, consequently, the uniform results indicated that there was no difference in finish between groups.

In order to make the final weights comparable, the weight data was plotted graphically and the theoretical value for pens 12, 13, and 14 extrapolated for June 11 when the cattle in pen 11 were last weighed.

Data is presented pertaining to the average worm counts and the percentage efficiency of the treatments (table 4), and also to the generic and specific percentile composition of the nematode populations (table 5). Had necropsy been performed at five to seven days following treatment, the results of worm counts would no doubt have been more meaningful; however, it is believed that the tabulated results are indicative.

In addition to those nematodes listed (table 5) from the abomasa, small numbers of less than 1 per cent of the population consisted of *Trichostrongylus axei* males, *Trichostrongylus* spp. females, *Cooperia* spp. females, and *Ostertagia circumcincta* males. In the small intestines, additional species encountered in numbers equivalent to 1 per cent or less were *Nematodirus spathiger*, *Ostertagia ostertagi*, and *Trichostrongylus* spp.

DISCUSSION

It is apparent that neither of the phenothiazine preparations incorporated in feed could be considered palatable since, in both cases, the feed consumption was markedly reduced.

Observations with regard to toxicity and phenothiazine absorption were necessarily superficial; however, no indication of toxicosis was seen in any group. It has been stated⁶ that, since the feces of cattle treated with carob flour-phenothiazine mixture turned red, less phenothiazine was

TABLE 4—Average Worm Count per Animal at Time of Slaughter and the Percentage Efficiency of the Treatment

Pen	Abomasum		Small intestine		Total	
	No.	%	No.	%	No.	%
11	177	44	159	17	336	34
12	225	29	152	20	337	26
13	316	—	191	—	507	—
14	122	61	150	21	272	46

absorbed; the fallacy of this has been pointed out.⁷ The degree of discoloration of hair contaminated with urine is considered a much better, although still inadequate, indication of phenothiazine absorption.

The conclusion based on the intensity of preputial hair discoloration is that there was no evidence indicating a difference in total absorption of phenothiazine in any of the treated groups. The variation in intensity of preputial hair discoloration between animals in groups 11 and 12, in contrast to those in pen 14, indicates that feeding of the drug, even over a three-day period, results in a markedly irregular dosage rate from animal to animal.

The small difference in weight gains between groups would indicate that treatment of such animals by any of the methods used is not an economical practice.

The fact that no increased weight gains were obtained from treatment could be due to one, or both, of two possibilities: (1) As indicated by the low parasite egg counts, there may have been insufficient numbers of parasites to interfere with the growth rate of the steers; and (2) the treatments did not remove a sufficient number of parasites to allow the treated animals to gain more than the control animals.

Statistical analyses (t-test for significance of difference between means) of total worm counts from individual animals (table 4) revealed that, in the small intestines, the differences between groups are not significant and could be due to chance.

TABLE 5—Generic and Specific Percentile Composition of Worm Populations

Nematode	Pen 11			Pen 12			Pen 13			Pen 14		
	Species	Genus		Species	Genus		Species	Genus		Species	Genus	
Abomasum												
<i>Ostertagia ostertagi</i>	♂	33	97*	24	100		31	99*		31	99*	
<i>Ostertagia</i> sp.	♀	64	—	76	—		68	—		68	—	
Small intestine												
<i>Cooperia oncophora</i>	♂	28	—	22	—		29	—		30	—	
<i>Cooperia mc masteri</i>	♂	3	92*	3	95*		—	97*		2	96*	
<i>Cooperia</i> sp.	♀	61	—	70	—		68	—		64	—	
<i>Nematodirus filicollis</i>		2	—	3	—		1	—		2	—	

*See text for difference between this and expected 100 per cent.

Analyses of data on the worm counts from the abomasas revealed that the difference between the drenched animals (pen 14) and the controls (pen 13) was highly significant, $P = \text{less than } 0.01$. The difference between animals treated with the carob flour-phenothiazine preparation (pen 11) and the controls (pen 13) was found to be significant, $P = \text{less than } 0.05$, but more than 0.02 . No other differences were significant. In addition, there appeared to be no differential effect between species (table 5).

Since work with sheep³ has shown that a significant decrease in anthelmintic efficiency results when the therapeutic dose of phenothiazine is divided into three or more daily doses, and there is every reason to believe the same results would be obtained in cattle, the apparent superiority of the carob-flour preparation over the flavored liquid preparation would seem to deserve some explanation.

Because of this, the size of the particles of phenothiazine used in preparing the carob flour-phenothiazine material employed in this trial was determined.† Information supplied by the producer indicated that the mean particle diameter was approximately 10.0μ ; however, analysis by the Fisher sub sieve air permeability apparatus revealed it to be 3.5μ .

If in cattle, as in sheep,^{2,4} there is a linear relationship between specific surface, a function of particle size, and anthelmintic efficiency of phenothiazine, this could well account for the fact that the carob flour-phenothiazine preparation used in this trial and fed for three days was as efficient as a single therapeutic treatment with commercial N.F. drench in which the particles averaged 10.0 to 13.0μ in diameter. It therefore follows that, if a drench were prepared from the same phenothiazine as used in preparation of the carob flour mixture, the drench would be expected to give a significantly higher efficiency.

SUMMARY

1) A flavored phenothiazine liquid and a carob flour-phenothiazine mixture used in the present trial were both found to be unpalatable to feedlot cattle.

2) The treatment of feedlot cattle with phenothiazine under the conditions of this trial was not an economical practice.

3) No signs of toxicosis were observed in any treated animals.

†Determination was made in the Department of Engineering, University of California, Berkeley.

4) Limited observations indicated that the total amounts of phenothiazine absorbed by animals in each treated group were similar; however, administration of phenothiazine in feed, even over a three-day period, apparently results in gross inequality of dosage among individual animals.

5) Treatment of cattle with carob flour-phenothiazine mixture was no more effective than drench preparations when used as in the present trial and, if phenothiazine of similar particle size were used in both preparations, the drench would undoubtedly be superior.

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Phenothiazine—Particle Size and Toxicity.—In an experiment with lambs, phenothiazine of small particle size (which is more efficacious as a vermifuge) was no more toxic than the coarser product.—P. A. Kingsbury in Vet. Rec. (June 28, 1958): 523.

Induced Trichinellosis in Swine

Fifty-five pigs, 2 months to 8 years old, were fed encysted larvae of *Trichinella spiralis* in ground pork, 400 to 600 larvae per pound of body weight.

When killed, 21 to 162 days later, *Trichinella* were recovered from the following tissues (percentage given in parentheses): diaphragm (100); stomach wall (18); testes (15); liver (11); brain, lung, and wall of the small intestine (9); pancreas and aorta (8); urinary bladder contents (7); urinary bladder wall (5); and heart (2).—Vet. Bull. (April, 1958): Item 1110.

The Failure of Egg-Propagated Distemper Virus to Interfere with Experimental Neurotropic Distemper in Mink

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IT HAS BEEN reported that egg-adapted distemper virus (DV), administered intracerebrally to dogs in the early stages of distemper, would prevent neurological signs.² The present paper deals with an attempt to prevent clinical distemper encephalitis in mink with a modified virus, given by intracerebral injection following virulent DV exposure.

MATERIALS AND METHODS

Virus Suspensions.—Green's ferret-passaged agent¹ has been maintained in this laboratory by occasional passage of frozen spleen suspensions and lyophilized samples through ferrets. It is pathogenic and encephalitogenic for young mink.

The Onderstepoort egg-adapted virus is an egg-adapted variant of Green's virus¹ and was employed in this test at the ninetieth chorioallantoic membrane passage. After trituration and short centrifugation at 2,000 r.p.m., the supernatant fluids were sealed in glass and stored at -20 C. until used. The diluent consisted of nutrient broth (Difco) containing 500 units of penicillin and 100 µg. of streptomycin per milliliter.

ocular and nasal exudate were considered catarrhal signs. Intermittent convulsions were recorded as nervous signs. The legs and bodies became rigid, and the jaws champed and jerked, with saliva appearing at the corners of the mouths. Piercing screams were common during the attacks. Distemper inclusion bodies were demonstrated in the urinary bladders of animals dying early in the disease.

Mink.—The animals (standard dark and mutant color phases) used in this test included both males and females. They were all at least 10 weeks of age in order to exclude maternal antibody as a factor in modifying the experimental disease. The regular stock ration was fed. Each mink was housed in an individual pen.

EXPERIMENTAL PROCEDURE

Sixty-nine young mink were given 1 ml. of a 10^{-1} suspension of Green's virus subcutaneously. Nine days later, 37 mink were selected at random and given 0.5 ml. (10^{-1}) of egg-propagated virus by the intracerebral route. The remaining 32 mink served as untreated controls. The results (table 1)

TABLE 1—The Results of Intracerebral "Treatment" of Distemper in Mink, Using Egg-Adapted Distemper Virus

No. of mink	Days between exposure and treatment	No. showing catarrhal signs	No. showing nervous signs	Deaths from distemper	No. surviving
37	9	36 (97%)	31 (83%)	37 (100%)	0
32	Controls— not treated	29 (91%)	25 (78%)	31 (97%)	1

Technique of Intracerebral Inoculation.—The mink were anesthetized with ether. The inoculation sites were shaved, cleansed with green soap and water, and painted with tincture of mercuric iodine. A hole was drilled through the skulls over the center of the right cerebral hemisphere. In this operation, a Steinman pin was used with a special device* to secure the skin, fascia, and muscles to the skull in a fixed position.

Criterion of Infection.—Serous or mucopurulent

indicate that the "interfering virus" had no effect in altering the course of disease in respect to either encephalitic or catarrhal signs. The animals were observed for 60 days.

DISCUSSION

Egg-passaged DV does interfere with the lethal action of Green's virus in ferrets. In all such cases, however, the interfering egg virus preceded the excluded virulent agent. Simultaneous injection of opposing viruses, or virulent virus given prior to the egg-passaged virus, failed to protect ferrets.¹ Ferret-adapted distemper virus, producing only a mild disease in silver foxes,

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*A description of this device will be presented in a separate report.

when injected simultaneously with, or as late as 12 days after, intranasal exposure of the foxes to virus fully virulent for this species, prevented the fatal disease.⁵

If neurotropic signs as a manifestation of virulent virus multiplication are to be excluded, the blocking virus—in this case, egg-passaged DV—probably should be given within a relatively short interval after the virulent virus. The interval of nine days is apparently too long.

Moreover, it has been shown² that the virus titer in the brain of mink on the eighth day after exposure was at least 10^{-2} . On the tenth day, the recorded titer was 10^{-3} . Without belaboring the point, it is felt that such evidence precludes administration of egg-adapted virus late in the incubation period.

The nine-day interval was chosen because it is about the length of the incubation period before clinical signs first appear in mink. Furthermore, it roughly corresponds to the time elapsed before a practitioner is first presented with a dog infected with distemper.

A few clinical trials on distemper cases in dogs at the Washington State College veterinary clinic have failed to verify the findings of others. Alternate cases which were diagnosed as early clinical distemper (with its inherent limitations) were treated. The eventual outcome was similar in both groups, in that about 75 per cent of those in the test showed neurotropic signs.

SUMMARY

Egg-propagated distemper virus given to mink by the intracerebral route, nine days after subcutaneous virulent virus exposure, did not interfere with the production of neurotropic signs or mortality.

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Control of Lice in Range Cattle

The period of residual effectiveness of several agents for louse control was determined for six months after a single application of the wettable powder suspensions. Chlordane (0.5%), heptachlor (0.06%), dieldrin (0.03%), and malathion (0.5%) each afforded protection for four to five months. Heptachlor provided the cheapest treatment. Several combinations of these drugs did not prolong the control. Lindane and perthane were inferior to the other insecticides. Only two products killed louse eggs: 52 per cent with malathion, and a small percentage with benzene hexachloride (0.025% gamma isomer).—*Vet. Bull. (May, 1958): Item 1469.*

Persistence of Aldrin and Dieldrin in Wool.—Sheep dipped in 0.05 per cent of aldrin or dieldrin were protected against fly strike for 12 to 40 weeks. These agents could not be removed from the fleece by scouring it with soap and sodium carbonate. There seems to be a combination between the wool fiber and the agent which resists solvent but leaves the insecticide able to kill larvae moving over the fiber surface.—*Nature (May 3, 1958): 1267.*

Muscle Changes in Scrapie.—The skeletal muscles from 30 sheep affected with scrapie, 14 experimental and 16 natural cases, were compared with those of 12 normal control sheep. Macroscopic lesions were found in 1 sheep with natural infection, and minor microscopic changes in 5 (2 natural, 3 experimental). It was concluded that scrapie can not be considered a primary disease of the muscles and that muscle changes play no apparent part in producing clinical signs of the disease.—*J. Comp. Path. & Therap. (April, 1958): 284.*

Correction—Blood of Race Horses

In the articles "The Blood Picture in the Race Horse. I" by C. H. G. Irvine, and "Further Erythrocyte and Hemoglobin Studies in Thoroughbred Racing Horses" by H. C. Brenon, in the July, 1958, *JOURNAL* (pp. 97-104), the measurement for erythrocytes should be million/cmm.; and in the latter article, the measurement for PCV should be per cent, not mm.

First Report of the Occurrence of Neonatal Endocardial Fibroelastosis in Cats and Dogs

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WHILE IT IS quite likely that congenital cardiac defects are as common in domestic animals as in man, few such cases have been reported—probably because of the infrequency with which neonatal deaths in animals are investigated by necropsy.

On the other hand, the acquired cardiac diseases common in man, such as coronary arteriosclerosis and acute coronary occlusion, are known to be rare in other animals.

Conversely, the common cardiac diseases of animals, such as bacterial endocarditis, myocarditis, myocardial necrosis secondary to vitamin deficiency, acute cardiac dilatation, and aortic rupture, are rarely seen in man.

It is, therefore, quite unusual to find a "human" disease of the neonatal period occurring spontaneously in a kitten or pup.

The 2 cases of endocardial fibroelastosis reported here are of interest because this disease has never before been reported in these species. The only case report for animals is a description of the disease in cattle.¹¹

CASE REPORTS

Case 1.—This 2-month-old, male Siamese cat was the only one of two litters of kittens to reach weaning age. All of the 8 kittens appeared normal at birth but matured slowly and, in turn, stopped nursing, became quiet, and finally died after about two days of illness.

Unfortunately, the owner disposed of all but 2 of the kittens without having them examined. One kitten, that died at 2 weeks of age, showed an enlarged heart but was not examined histologically. The other kitten was always stronger than the other

7 but its illness followed a similar, although slower course, and it died at 2 months of age. The owner decided that this condition was hereditary and had the mother cat spayed before histological examination of the kitten's heart was completed. Thus the possibility of obtaining additional litters for further investigation was lost.

At necropsy, in addition to severe dehydration, there was pulmonary edema and cardiac dilatation and hypertrophy. This heart was similar to one seen later in a dog (case 2) and that seen previously in the sibling of this kitten.

While all the cardiac chambers were dilated, the dilatation and hypertrophy of the left ventricle was most extreme. The endocardium of the left ventricle was thickened and opaque, obscuring the usual muscular markings (fig. 1B). Partial aortic stenosis was present.

Microscopic examination revealed unusual fibroelastosis of the endocardium and myocardial hypertrophy (fig. 2B).

Case 2.—This 4-month-old female Standard Poodle was purchased when about 11 weeks old. She was small for the breed, did not play spontaneously, but preferred to lie around. Five days after purchase, she stopped eating and rapid respirations developed.

Two days later, she was brought to the veterinary clinic, where dyspnea and enlarged, hyperemic tonsils were the only abnormalities found. Although the body temperature and thoracic sounds were normal, an antibiotic was given parenterally.

On the following day, the heart beat was rapid and pounding, respiration was labored, the mucous membranes were pale, and the animal refused to move about. A diagnosis of cardiac failure due to an unknown cause was made.

Because of the age of the dog and the consequent poor prognosis, digitalization was not attempted. The dog became comatose that evening and died the next morning, the ninth day after purchase.

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This work was done under the auspices of the AEC.

The authors express their appreciation for aid in this study afforded by Mrs. Gretchen Humason in the histological preparations, Mrs. Julia M. Wellnitz in photography, and Mrs. Betty Linke in preparing the manuscript.

At necropsy, the heart (fig. 1C, D) was about three times the volume of the normal heart of a pup of this age and breed. Much of the cardiac enlargement was due to the enormous dilatation of all the chambers, but particularly that of the left ventricle and atrium. There was also hypertrophy of the left ventricular wall.

The endocardium of the left ventricle was thickened and opaque so that it obscured the usual trabeculae carneae. Partial aortic stenosis was present in this case also.

The only other abnormalities found were enlarged tonsils, hypostatic bronchopneumonia, and hepatic fatty changes.

Microscopically, the myocardial muscle fibers showed hypertrophy and bizarre enlargement of their nuclei, as commonly encountered in diseases causing enlargement of the human heart.

There was only a slight increase in the amount of interstitial myocardial connective tissue. No alteration of the capillaries and small blood vessels nourishing the myocardium and no acute inflammatory changes were present.

Extreme endocardial thickening was due to an increase in collagenous connective tissue. Verhoeff's stain for elastic tissue revealed an unusual proliferation and hypertrophy of the endocardial elastic fibers in this region (fig. 2C, D). This change was so extreme that, in some areas, the endocardium of the left ventricle, which in the newborn is normally thin and contains only a few tenuous elastic fibers, actually had the histological appearance of the aorta.

Microscopic examination of the other organs failed to contribute any possible explanation for the cardiac abnormality.

DISCUSSION

Endocardial fibroelastosis is still considered a congenital disease of unknown cause by most pathologists. Originally it was called fetal endocarditis^{9,12} because it was thought to be the result of an intra-uterine infection. As this supposed infectious origin became increasingly doubtful, the disease was given the descriptive name it now bears.^{9,13}

As a result of the obscurity of its origin,

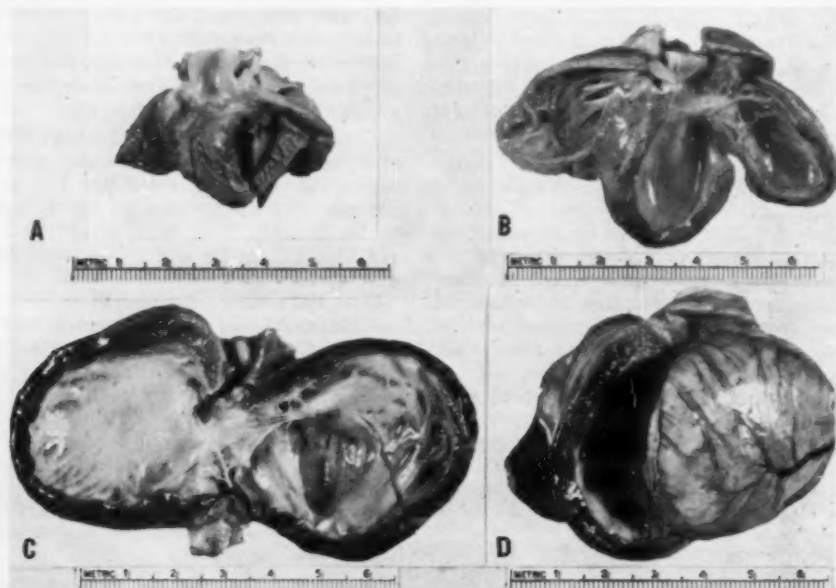


Fig. 1—Gross appearance of the hearts of a dog and cat (about natural size). (A) Heart of a normal kitten about 2 months of age; (B) heart of a kitten the same age with neonatal endocardial fibroelastosis (case 1)—notice dilatation of the chambers, myocardial hypertrophy, opaque endocardial thickening of the left ventricle, and aortic stenosis; (C, D) heart of a dog with neonatal endocardial fibroelastosis (case 2)—notice the obvious hypertrophy, dilatation, and opaque endocardial thickening as in B.

there is much literature on this disease, in human beings, which has recently been reviewed.^{1,2,5,7}

Some of the etiological concepts that have been formulated are: familial inheritance of a morphological or developmental

defect; heterotopic aortic tissue in the ventricle; hyperplasia of the aortic connective tissue with extension into the ventricle; hyperplasia of the subendocardial collagenous and elastic tissue as a response to inadequate myocardial contractility; un-

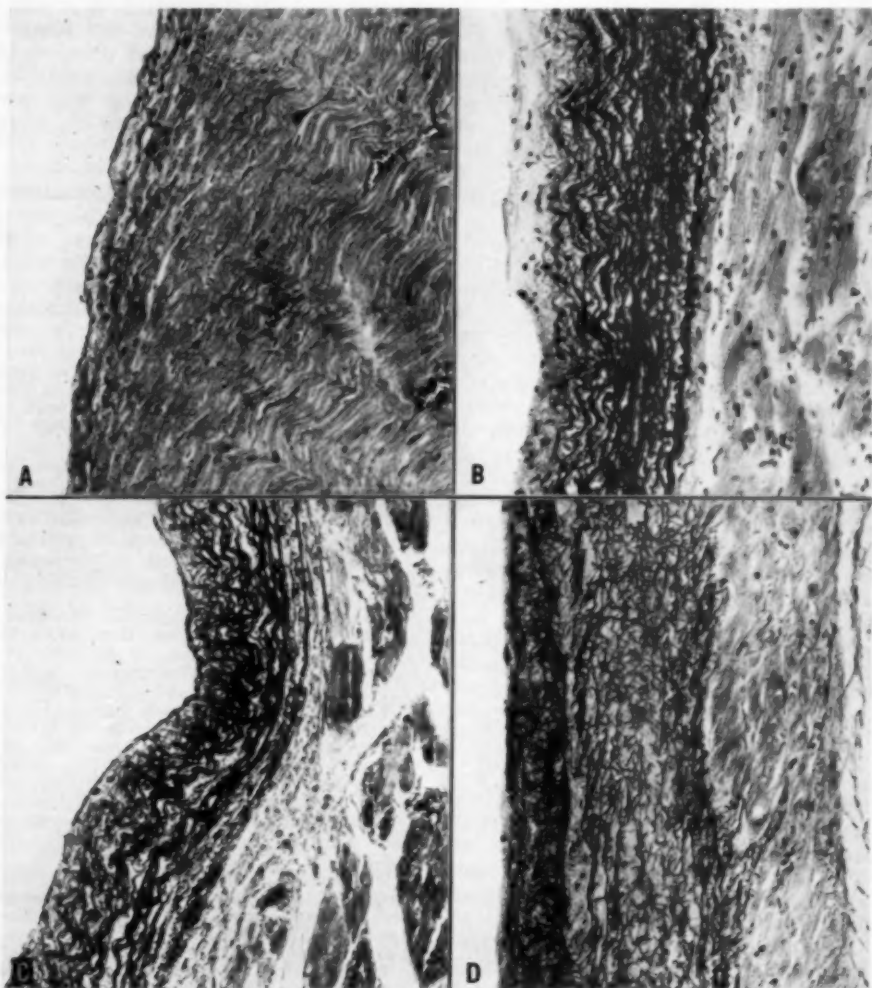


Fig. 2—Microscopic appearance of left ventricle and endocardium. x 225. Sections taken parallel to the long axis of the heart through the anterior portion of the ventricular muscle, about halfway between the valve ring and the apex, stained by Verhoeff's method for elastic fibers (preferentially stained black), showing the endocardial surface on the left, and the underlying myocardium of the left ventricle on the right.

(A) Section from a normal kitten—notice thin endocardium which at this magnification is not easily discernible; (B) section from abnormal kitten (case 1)—notice thickness of endocardium, density of black staining elastic fibers, and hypertrophied myocardial fibers; (C) section from abnormal pup (case 2)—same as B; (D) section from abnormal human infant with "congenital" endocardial fibroelastosis—notice that the only difference between the human and animal lesions is the greater diameter, and hence darker staining, of the elastic fibers of the animals.

known metabolic disorder of the cardiac muscle and connective tissue; a "collagen disease" of the heart; a response to anoxia from aberrant pulmonary coronary arteries; premature closure of the foramen ovale or other valvular or vascular anomaly; and a hyperplastic response to anoxemia and prematurity.⁶

Although a detailed discussion of the etiological factors in endocardial fibroelastosis is beyond the scope of this paper, it appears that this is not a disease *sui generis*,³ but a condition which can result from many factors, both hereditary and acquired. A congenital hereditary factor is probably most important in the "idiopathic" cases.

The uniformity of the clinical course^{4,10} of the 7 siblings of the kitten in case 1 suggests that a familial, hereditary factor was present and active here. The neonatal state of the 2 animals in this report and the numerous case reports on the disease in man indicate that this condition has a congenital inception.

The presence of aortic stenosis in the 2 animals in this report would seem to support the anoxic etiological concept, which states that the endocardial fibroelastic hyperplasia is a reaction to myocardial hypertrophy and weakness resulting from relative ischemic anoxia of the myocardium, caused by an abnormal intra-uterine cardiac circulation.

Myocardial anoxia of the left side of the heart appears to be produced more easily during intra-uterine life than after birth. This possibility is suggested by the fact that early in the development of the heart there is no coronary circulation and myocardial nourishment must be derived from the cavity blood. Premature closure of the foramen ovale would interfere with the flow of oxygenated fetal blood from the right atrium to the left atrium and cause myocardial and endocardial ischemia¹¹ and damage to the left side.

The resulting decreased flow of blood through the left side of the heart would also allow fusion of damaged endocardial surfaces of valve cusps, with the production of aortic stenosis. The aortic stenosis and mitral incompetence, thus established, would lead to further cavity anoxia and set into play the hyperplastic endocardial response and myocardial work hypertrophy which lead ultimately to myocardial failure and the typical death from this disease.²

The occurrence of this phenomenon in lower animals would seem to offer hope that such etiological speculations as this might ultimately be placed on the sound basis of experimental observation.

The occurrence of fibroelastosis in a short neonatal period, with a clearly defined clinical course,^{4,10} makes it a clinical disease recognizable before the death of the patient. These cases also illustrate the well-defined course of the disease in animals, which should enable more veterinarians to diagnose this disease in the future.

SUMMARY

Two cases of infantile endocardial fibroelastosis, 1 each in a cat and dog, are described for the first time. Some of the etiological concepts concerning this disease and some of its implications, both in research and clinical practice, are discussed.

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Simple Methods for the Detection of Unfavorable Changes in Ruminal Ingesta

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AT TIMES, it may be of value to practitioners to ascertain whether the microbial activity of the rumen is "healthy." Because of the complexity of the numerous biochemical processes associated with such activity, it would be impractical to even attempt any detailed analyses as mere aids to the diagnosis of indigestions. Nor can one count and classify organisms as a simple estimate of the over-all activity of the ingesta.

It is possible, however, to obtain a fairly reliable appraisal of normal activity by comparing what one sees and smells in a sample of ruminal ingesta obtained by stomach tube with what one has seen and smelled previously in ingesta from healthy cows on similar rations.

However, with the present-day addition of many compounds and drugs to the feed, such simplified comparisons need fortification with additional practical methods of measuring the activity of the ingesta. In addition to measurements of pH,² there are two procedures which offer promise: One is called the sediment activity test; the other the cellulose digestion test. The latter is a modification of Moore's procedure.³ Both can be accomplished with samples taken by stomach tube.

SEDIMENT ACTIVITY TEST

Rumen fluid is obtained and strained immediately through one layer of gauze or cheesecloth to remove coarse particles. The strained portion is thoroughly mixed and poured into a suitable vessel and the time is recorded. The sample is then placed where it can be maintained without agitation at about body temperature.

As time progresses, it will be observed that the particulate material of the normal samples begins to clump and may start to settle (fig. 1, cylinder at the left). However, before much settling can take place, it will be observed that clumps of particulate material containing bubbles of



Fig. 1—The graduated cylinder on the left contains a fresh sample of strained ruminal contents, the particles of which are beginning to agglutinate. The particles in the center sample have been buoyed to the surface by the bubbles of gas produced from them by the gas-producing ruminal organisms. The particles of the sample on the right have settled because normal bubble formation has been reduced markedly.

gas reverse their direction and begin to float to the surface.

The time in minutes that intervenes between the filling of the cylinder and completion of the floating of the mass of particles (fig. 1, center cylinder) is recorded as the sediment activity time (SAT).

Duplicate measurements of the SAT of five replicate samples, taken 30 minutes after eating from similar regions of the rumens of 2 fistulated cows being fed alfalfa hay, indicate a reasonable reproducibility of sampling technique (table 1).

Coefficients of variation (5, 16, 12, 5, 6, 4, 7, 6, and 0%) of ten sample replicates of each of nine different samples of ruminal

TABLE 1—Reproducibility of Sediment Activity Times of Five Duplicate Samples from the Same Region of the Rumens of Two Cows

Sample No.	Sediment activity time (min.)			
	Cow 1		Cow 2	
1	3.0	3.0	3.0	3.0
2	3.0	3.0	4.0	4.0
3	2.0	2.0	2.0	2.0
4	3.0	3.0	3.0	3.0
5	2.5	2.5	2.0	2.0
Means	2.7	2.7	2.8	2.8

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TABLE 2—Effects of Eating and Watering upon Sediment Activity Time (SAT) of the Same Cow Fed Two Different Feeds

	Before a.m. feeding	Feed	Interval after feeding				
			15 min.	30 min.	1 hr.	2 hr.	5 hr.
No. of samples	12	Alfalfa hay	75	50	60	Water	12
SAT (min.)	7.2		3.3*	4.3*	5.5		4.3*
No. of samples	19	Fresh ludino	22	22	20	Water	22
SAT (min.)	8.7		3.9*	3.7*	4.1*		3.6*

*Significant at the 1 per cent level.

ingesta indicate a reasonable reproducibility of results of similar portions of the same sample.

Patterns of changes in the SAT are associated with eating and drinking (table 2). There are also variations between cows, and in the same cow from day to day (table 3).

The normal gas activity may be materially interfered with by many agents administered or fed to the animal, as well as

by starvation, inappetence, and indigestions. This is reflected either by delays in the floating of the particulate material, by incomplete floating, or by complete settling without any floating. Serious interference results in the latter (fig. 1, cylinder at the right). Restoration of gas activity is evidenced by a return to normal SAT. Periods of excessive frothing induced by feeding bloat-provoking legumes are accompanied by periods of more rapid SAT.

CELLULOSE DIGESTION TEST

A sample of strained ruminal fluid is obtained as described. Ten milliliters is transferred to a screw-capped 15-ml. test tube. A single strand of unmercerized cotton thread, weighted at one end by a large bead or a short length of glass tubing tied to the thread, is suspended in the ruminal fluid and held in suspension by screwing the cap of the test tube over the upper end of the thread (fig. 2). The sample is then maintained without agitation at about body temperature either in an incubator or near a light bulb.

The tube is examined in 24 hours. If the thread has broken, the bead will be seen at the bottom of the tube. If the thread has not broken, the tube is incubated for another six hours. If the thread has not broken in 30 hours, results of several hundreds of determinations suggest that cellulose digestion time (CDT) should be interpreted as being delayed.

Feeding and watering times have less effect on CDT than on SAT but, as with SAT, the feeding or administering of many agents, as well as many kinds of indigestions, are accompanied by either delay or cessation of activity, and a resumption of a normal CDT indicates a return to normal.

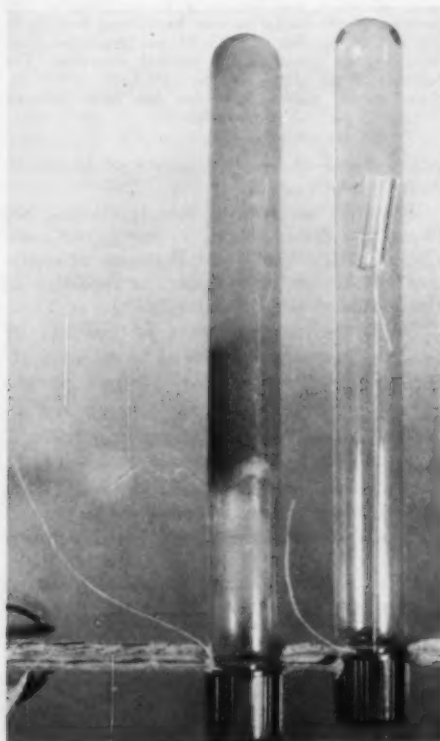


Fig. 2—Equipment used to expose a weighted cellulose thread to a sample of ruminal contents.

TABLE 3—Variations in Sediment Activity Times Between Cows and in the Same Cow on Different Days

Day	Before a.m. feeding		Interval after feeding									
			15 min.		30 min.		1 hr.		2 hr.		5 hr.	
	Cow 1	Cow 2	Cow 1	Cow 2	Cow 1	Cow 2	Cow 1	Cow 2	Cow 1	Cow 2	Cow 1	Cow 2
1	9.0	4.0	2.5	2.5	5.0	4.0	3.0	4.0	4.0	4.0	4.0	5.0
2	3.5	14.0	2.0	7.0	2.0	5.0	2.0	4.0	2.0	9.0	2.5	2.5
3	2.0	3.0	2.0	2.0	2.0	2.0	3.0	3.0	3.0	3.0	4.0	3.0
4	3.0	6.0	3.0	4.0	4.0	5.0	2.0	3.0	2.0	6.0	3.0	3.0
5	5.5	3.0	3.0	3.5	3.0	4.0	3.5	6.0	3.0	3.0	3.0	7.0
6	13.0	19.0	5.0	6.0	4.0	6.0	10.0	4.0	4.5	4.0	5.0	4.0
Means	6.0	8.2	2.9	4.2	3.3	4.3	3.9	4.0	3.1	4.8	3.6	4.0
	7.1		3.5		3.8		4.0		4.0		3.8	
1	8.0	12.0	4.0	4.0	5.0	3.5	3.0	3.0	2.5	4.5	5.0	4.0
2	5.0	7.0	3.0	4.0	3.0	3.0	4.0	8.0	3.0	6.0	3.0	4.0
3	1.0	4.0	2.0	2.0	2.0	2.0	2.0	2.0	2.0	3.0	2.0	2.0
4	6.0	7.0	3.0	3.0	2.0	4.0	3.0	4.0	2.0	4.0	2.0	3.0
5	8.0	3.0	4.0	5.0	3.5	5.0	4.0	5.0	4.0	4.0	4.0	3.5
6	11.0	11.0	6.0	6.0	6.0	6.0	5.0	6.0	4.0	7.0	4.0	7.0
Means	6.2	7.3	3.7	4.0	3.6	4.3	3.5	3.7	2.9	4.8	3.3	3.9
	6.8		3.8		3.9		4.1		3.9		3.6	

For example, the administration, with grain, of 75 mg. of penicillin produces a temporary delay in CDT, without materially affecting SAT (table 4).

TABLE 4—Effects of Feeding a Single 75-mg. Quantity of Penicillin on Cellulose Digestion Time (CDT) and Sediment Activity Time (SAT)

Time sample taken	CDT (Time for thread to break)	SAT
Before a.m. feeding	26 hr.	13 min.
Interval after feeding penicillin:		
30 min.	Not broken in 4 days	3 min.
1 hr.	Not broken in 4 days	4 min.
2 hr.	Not broken in 4 days	2 min.
3 hr.	Not broken in 4 days	2 min.
5 hr.	3 days	5 min.
24 hr. (before feed—2nd day)	26+ hr.	5 min.

It is possible to conduct both tests upon the sample contained in the capped test tube used for the CDT.

DISCUSSION AND CONCLUSION

Simple as they may be, the above two tests allow a reasonable appraisal of the activity of the gas-producing and cellulose-digesting organisms of the rumen, particularly if one uses them frequently enough to acquire the necessary experience and caution for judicious interpretation. They are sensitive enough to reveal even minor or transitory delays in rates of digestion.

From a research standpoint, they have been valuable tools for screening compounds which do or do not interfere with these types of digestion.

Their use in this respect also indicates that compounds to be tested for toxicity, when given orally to ruminants, must first be examined for their effects upon the function of ruminal organisms. If this is not done, it is possible that a compound which produces illness in an animal is interpreted as an organic poison to the animal when, in reality, it may be producing its major effects by depression of the activity of the digestive microorganisms. Such compounds may not necessarily be harmful if used parenterally rather than orally.

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Mineralized Artesian Water and Pigs

Hogs given drinking water containing 2.5 to 3.5 Gm. per liter of chlorides and 500 mg. per liter of sulfate showed no ill effects.

The excessive chlorides were excreted in the urine. However, this water was harmful to unweaned and recently weaned pigs, causing digestive disturbances, skin rash with itching, and a reduced growth rate.—*Vet. Bull. (May, 1958): Item 1506.*

Ethical Promotion of Drugs

A recently published statement (*J. Am. Pharm. A.*, July, 1958: 409) of principles adopted by the Pharmaceutical Manufacturers' Association regarding the ethical promotion of pharmaceutical products to the medical and allied professions is worthy of notice. The text of the statement follows:

We, members of the Pharmaceutical Manufacturers' Association, recognizing our responsibilities and obligations to promote the public welfare and to maintain honorable, fair, and friendly relations with the medical profession, with associated sciences, and with the public, do pledge ourselves to the following statement of principles:

1—Prompt, complete, conservative and accurate information concerning therapeutic agents shall be made available to the medical profession.

2—Any statement involved in product promotional communications must be supported by adequate and acceptable scientific evidence. Claims must not be stronger than such evidence warrants. Every effort must be made to avoid ambiguity and implied endorsement. Whenever market, statistical, or background information or references to unpublished literature or observations are used in promotional literature, the source must be available to the physician upon request.

3—Quotations from the medical literature or from the personal communications of clinical investigators in promotional communications must not change or distort the true meaning of the author.

4—If it is necessary to include comparisons of drugs in promotional communications, such comparisons must be used only when they are constructive to the physician and made on a sound, professional and factual basis. Trademarks are private property that can be used legally only by or with the consent of owners of trademarks.

5—The release to the lay public of information on the clinical use of a new drug or a new use of an established drug prior to adequate clinical acceptance and presentation to the medical profession is not in the best interests of the medical profession or the layman.

6—All medical claims and assertions contained in promotional communications should have medical review prior to their release.

7—Any violation of these principles brought to the attention of the president of the Pharmaceutical Manufacturers' Association shall be referred by him to the Board of Directors.

Veterinarians will approve this progressive step taken by pharmacists.

The above statement of principles does

not pertain to drugs advertised directly to the public, nor to many products such as antibiotics available for the treatment of livestock diseases. The distinction between drugs designated for use by veterinarians only and for sale to owners of animals is not as clear as is the distinction between drugs for prescription use for man and for over the counter sales.

PREMATURE RELEASE OF DRUGS

About six years ago a synthetic hormone, which was claimed to effect "market-matched breeding" of ewes and to result in a uniform lamb crop, was extensively advertised in veterinary publications and in farm magazines. Had the claims been justified, use of this product would have been a boon to the sheep industry. The ewes in a flock could have been induced to produce more lambs, in a shorter lambing period and to raise two crops of lambs a year. Therefore, many sheep breeders hopefully purchased and used the drug. Little did they suspect that the induced estrus would not be accompanied by ovulation.

When two experiments, reported independently, showed that pregnancies did not result from use of the drug, the advertising in domestic publications was promptly withdrawn; however, it continued for some time in at least one foreign English-language publication.

This was not the first time that a manufacturer's claim for a product has been contradicted by field trials nor will it be the last. The question is, What should be done about it? Most manufacturers, as well as members of the profession, would like to establish safeguards against the premature release of drugs, but how can this best be accomplished? Should organized veterinary medicine establish testing facilities and either grant or withhold a stamp of approval on tested drugs? Should existing state institutions such as colleges and agricultural experiment stations undertake the task? Should an organization of manufacturers do it? Could it be done through cooperative effort?

With the marked acceleration in the introduction of synthetic and other drugs, the need for a clarification of principles regarding promotion and use of veterinary drugs will increase.

ABSTRACTS

Metabolic Patterns of *Brucella* Species

The comparative metabolism of resting cells of the three species of *Brucella* was determined on substrates of amino acids, carbohydrates, and Krebs' cycle intermediates. Quantitative and qualitative differences between the species were observed, particularly on amino acid substrates. The metabolism of each species was characteristic and distinctive, and differentiation of the organisms within this genus can be achieved by determining their basic metabolic pattern.—[Margaret E. Meyer and H. S. Cameron: *Species Metabolic Patterns Within the Genus Brucella*. *Am. J. Vet. Res.*, 19, (July, 1958): 754-758.]

Surgical Technique for Forming Bladder Pouches

A technique for the formation of bilateral isolated pouches of the canine urinary bladder was described and illustrated. The enclosed mucosa remained histologically normal for periods of at least three months after such isolation. Intravenous urography was used to determine whether the pouches were completely isolated from the bladder proper.

Such pouches should be suitable sites for tumor transplants, investigations of acute or chronic effects on the mucosa of substances introduced into the pouch, and for investigating the comparative effects on the urinary epithelium of substances excreted in the urine.—[Thomas B. Clarkson, William H. Boyce, and J. Stanton King, Jr.: *A Surgical Technique for the Formation of Isolated Pouches of the Canine Bladder*. *Am. J. Vet. Res.*, 19, (July, 1958): 661-665.]

Isolates of Avian Infectious Bronchitis Virus

Isolates (14) of avian infectious bronchitis virus (IBV) were studied by reciprocal serum-neutralization (SN) tests, in an attempt to demonstrate antigenic differences. An additional IBV isolate was tested against 14 prepared antisera. Three isolates were found to be immunologically distinct; two of these were considered strains of avian IBV because of their similarity to other isolates in pathogenicity for chickens and embryonating chicken eggs. One isolate requires further study for its classification. The others were found to be antigenically related, but there was considerable variation in the neutralizing indexes.—[M. S. Hofstad: *Antigenic Differences Among Isolates of Avian Infectious Bronchitis Virus*. *Am. J. Vet. Res.*, 19, (July, 1958): 740-743.]

Blood and Urine Constituents of Ewes

Ewes in which ketonemia and hypoglycemia had been induced previously, by fasting and phlorhizin treatment, were given acetic, propionic, or butyric acid orally.

Acetic acid moderately increased blood ketone levels above those of water-treated control animals, but did not significantly alter blood glucose or urinary urea nitrogen levels. The degree of the phlorhizin-induced glycosuria also was unchanged.

Propionic acid markedly increased the blood glucose levels and the degree of glycosuria and significantly reduced blood ketone levels.

Butyric acid significantly depressed blood glucose levels and reduced urinary glucose levels. Blood ketone levels were markedly increased. Total nitrogen and urea nitrogen excretion also were depressed.—[G. D. Goetsch and W. R. Pritchard: *Effects of Oral Administration of Short-Chain Fatty Acids on Certain Blood and Urine Constituents of Fasted, Phlorhizin-Treated Ewes*. *Am. J. Vet. Res.*, 19, (July, 1958): 637-641.]

Viability of Lyophilized Cultures

Cultures of 111 strains of bacteria, fungi, yeasts, and virus were suspended in Seitz-filtered equine serum and preserved by the lyophil process. Replicates of 53 cultures were stored for ten years at room temperature (approx. 22 to 32 C.) and at refrigerator temperature (approx. 6 to 7 C.). Fifty-eight cultures were stored at refrigerator temperature only.

With the exception of one strain of Newcastle disease virus, culture N₂ (N.J.-KD), there was no difference in survival at room temperature and at refrigerator temperature of cultures stored for ten years.

Lyophilized cultures of the turkey sinusitis agent, pseudorabies virus, and one strain of Newcastle disease virus, culture N₂ (Calif.-11914), did not survive storage for the period tested.—[Harry E. Rboades: *The Effect of Storage on Viability of Lyophilized Cultures of Bacteria, Viruses, Yeasts, and Molds*. *Am. J. Vet. Res.*, 19, (July, 1958): 765-768.]

Poultry Hemorrhagic Syndrome

Two lots of sterile grain on which one strain each of *Penicillium rubrum* and *Penicillium purpogenum* had been cultured were toxic for chicks; the former more so than the latter. Chicks showed depression, essentially no gain in body weight and, subsequently, died. Necropsy findings included hemorrhages and congestion of various tissues, erosion of the proventriculus and gizzard, and paleness of the bone marrow.

The addition of a protein-mineral-vitamin supplement to the fungal substrates resulted in less depression and fewer death losses due to mycotoxicosis. The addition of chlortetracycline to these rations resulted in fewer clinical signs and lesions of toxicosis.

One toxic strain each of *Aspergillus clavatus*, *Aspergillus flavus*, *P. purpogenum*, *P. rubrum*, *Paecilomyces variotii*, and a species of *Alternaria*, when inoculated into moist broiler mash containing no coccidiostat or antibiotic and incubated at ordinary room temperatures, produced toxic sub-

stances within six days and for at least 24 days. Necropsy findings in chicks that consumed the toxic mash were characteristic of the poultry hemorrhagic syndrome.

In a preliminary experiment, toxic fungi proliferated within 11 days in feed scattered by chicks in litter of wood shavings. Chicks maintained on such litter developed depression and diarrhea and, when the experiment ended at eight weeks, lesions of the hemorrhagic syndrome were found.

In a second similar experiment, fungal proliferation became evident on the eleventh day and the hemorrhagic syndrome was evident in chicks decapitated at 3½ weeks, reached its greatest intensity at 5 weeks, gradually decreased, and was absent at 12 weeks.

The presence of 200 p.p.m. of chlortetracycline in inoculated feed did not result in less fungal growth in feed scattered in litter of wood shavings, but there was less hemorrhagic syndrome in chicks maintained on such litter.—[J. Forgacs, H. Koch, W. T. Carl, and R. H. White-Stevens: *Additional Studies on the Relationship of Mycotoxins to the Poultry Hemorrhagic Syndrome*. *Am. J. Vet. Res.*, 19, (July, 1958): 744-753.]

Lesions of Virus Hepatitis in Ducks

Susceptible ducks were exposed orally and parenterally to a virulent and a chicken embryo-modified virus. The host reaction to the virus was studied in relation to microscopic tissue changes.

Significant lesions were observed in the liver and brain. Lesions in the liver consisted of necrosis of hepatic cells, perivascular infiltration with granulocytes and lymphocytes and, later, plasma cells and proliferation of bile duct epithelium. Perivascular infiltration, glial cell proliferation, and neuronal degeneration occurred in the brains of some of the infected ducks. The liver and brain lesions produced in the ducks were similar to those reported from cases of infectious hepatitis of man.

Administration of duck hepatitis immune serum to ducks 24 hours after exposure to the virus prevented further development of liver lesions. Although ducks given chicken embryo-modified virus developed liver lesions, no deaths resulted.—[L. E. Hanson: *Histological Lesions in Ducks with Virus Hepatitis*. *Am. J. Vet. Res.*, 19, (July, 1958): 712-718.]

The Hemograms of Healthy Chickens

Statistical analyses of avian hemograms indicated that a group of 20 to 30 experimental chickens would yield data on total leukocyte and erythrocyte counts that were as accurate as data collected from a group of 100 experimental birds.

A modification of the Guest-Siler microhematocrit method for blood cell volume determination, and a modification in the use of a Neubauer counting chamber for making total blood cell counts proved useful aids in the study of avian blood cells. Romanowsky-type stains revealed the pres-

ence of granular inclusions in the cytoplasm of immature heterophilic leukocytes and rod-shaped inclusions in the cytoplasm of more mature forms. The Romanowsky-type of stain was not satisfactory for distinguishing eosinophilic leukocytes from heterophilic leukocytes in avian blood.—[C. D. Diesem, W. G. Venzke, and E. N. Moore: *The Hemograms of Healthy Chickens*. *Am. J. Vet. Res.*, 19, (July, 1958): 719-724.]

Dissecting Aneurysms in Turkeys

Aortic ruptures caused by dissecting aneurysms were observed in turkeys in commercial turkey flocks in Indiana that had been fed rations high in proteins and fats. Fatal dissecting aneurysms also occurred in turkeys fed high protein and fat rations under controlled conditions. Turkeys fed rations high in protein and low in fat, or low in protein and high in fat, did not develop aneurysms.

Histological changes were observed in the aortic mediae, which were characterized by (a) an increase in ground substance which stained metachromatically with alcian blue, (b) fragmentation and disorientation of reticulum, (c) dissolution of the elastic fibers, and (d) fibroblastic proliferation. Intimal thickening and large fibrous intimal plaques composed of fibroblasts, connective tissue, and lipid consistently were found associated with ruptures of the blood vessels. Examination of blood vessels from the groups that did not develop fatal dissecting aneurysms during the six-month experimental period showed intimal changes, fibroblastic proliferative changes in the mediae, and dissolution of elastic fibers. These changes were identical to those of the survivors from the groups where fatal dissecting aneurysms had occurred.

Dissecting aneurysms and ruptures of the aortas, femoral arteries, and atria occurred in turkeys fed beta-aminopropionitrile (BAPN). No major histological differences could be detected between field cases of dissecting aneurysms, cases that occurred in turkeys fed high protein and fat rations under controlled conditions, and aneurysms that occurred in turkeys fed BAPN.—[W. R. Pritchard, Wilson Henderson, and C. W. Beall: *Experimental Production of Dissecting Aneurysms in Turkeys*. *Am. J. Vet. Res.*, 19, (July, 1958): 696-705.]

Bluecomb Disease of Turkeys

Preliminary studies indicate that parental immunity in day-old poults, obtained from breeding flocks recovered from bluecomb disease, either is lacking completely or is present at levels not measurable under the conditions of the experiments.

Antiserum, at a 1:2 dilution, from three flocks recovered from bluecomb disease, completely neutralized Sela 02 bacteria-free filtrates containing the infectious agent. Slight or no serum neutralization occurred at the 1:4 dilution for two flocks, but serum from flock 3 did partially neutralize the filtrate at the same dilution.

Weight gains of the uninoculated control poults

and of those inoculated with 1:2 serum-filtrate dilutions were consistently higher than those inoculated with infective filtrate alone or with 1:4 and 1:8 serum-filtrate dilutions.—[J. T. Tumlin and B. S. Pomeroy: *Bluecomb Disease of Turkeys. V. Preliminary Studies on Parental Immunity and Serum Neutralization. Am. J. Vet. Res.*, 19, (July, 1958): 725-728.]

BOOKS AND REPORTS

A Handbook of Animal Physiology

The author's objective was to provide a brief approach to various aspects of the subject and a framework of the main facts and scope of study of physiology and related disciplines. The book should be particularly valuable for the student beginning the study of biological science. Elementary discussions of the nervous system, including the autonomic system, and muscle are found in the first chapter. Respiration, digestion, metabolism, water and electrolyte balance, regulation of many important body functions, and reproduction are discussed.

Reference is made to many primitive forms of animal life as preliminary discussion for physiological processes in higher forms of mammals. The book is well illustrated and written in a manner so that it is easy to read. It is a book that should be of value to the beginning preveterinary medical student.—[*A Handbook of Animal Physiology*. By E. M. Paulclousis. 255 pages; illustrated. Bailliere, Tindall, and Cox, 7 and 8 Henrietta St., London, England. 1957. Price \$6.25.]—ROGER LINK.

Breeding Problems and Artificial Insemination

The author states that "This book emanates from a desire to make some contribution toward improving the fertility level of our animals. This work is therefore intended mainly for the use of stock owners, breeders, agricultural students, insemination technicians, and sterility workers in the hope that it will assist in improving the notoriously low breeding efficiency of our herds and flocks, and to raise the level of production as regards both quantity and quality."

General infectious diseases of the male and female genital tracts are discussed. The following pertinent observation is made relative to functional infertility. "On the surface of it one would consider that, since the immediate cause is an endocrine imbalance, the condition should readily respond to treatment with the appropriate hormone. On the contrary, the results obtained from hormonal therapy in the treatment of sterility are most disappointing in practice. In most cases, the mere injection of the relative hormone is fruitless unless the basic causes of the condition are first rectified."

One chapter is given to hereditary infertility, calling attention to the fact that genetic factors are of far greater importance in the production of

poor breeding efficiency in both males and females than is generally realized. Twelve abnormal conditions that can and often do cause sterility are briefly described.

In addition to the genetic influence upon infertility, the author discusses 16 lethal defects and 17 nonlethal deformities.

Part two is devoted to artificial insemination. It deals with the dangers associated with the practice and the methods of collecting and handling semen. Evaluation of the semen specimen and methods of injecting it into the cervix are described.

This is a good text. Veterinarians will find much of interest in it.—[*Breeding Problems and Artificial Insemination*. By S. W. J. van Rensburg. 250 pages; well illustrated. Labagric, Box 15, Pretoria, South Africa. 1958. Price not given.]—H. E. KINGMAN, Sr.

History of Veterinary Medicine

The history of veterinary medicine appears to be undergoing a revival in Europe after a considerable period of inattention. This Italian book is the third major historical work to be published in as many languages in the past few years, following that of Froehner in Germany and the revised version of Leclainche in French.

The present work contains a mine of information and is practically a bibliographic encyclopedia as well as a general history of veterinary medicine from the earliest times to the present. The contributions of more than 2,500 men are indexed, thus making the book a valuable reference work—although a number of important figures are given but brief notice, and there is no subject index. There is a 50-page section on the veterinary schools of the world.

A notable feature of the book is the considerable space given to the concurrent development of medicine and the allied sciences. This, however, results in some imbalance of subject matter; Darwin, for example, is accorded more space than is devoted to English veterinary medicine in either the seventeenth or eighteenth centuries—more than to the epochal work of Lancisi on the cattle plague in Italy. In fact, one might wish the author had used a more nationalistic approach; except for Ruini, relatively little space is accorded the important Italian authors of the Renaissance and later, when Italy was the chief agent in the regeneration of veterinary medicine in Europe. By comparison, the section on early veterinary medicine is treated fully and well, with many interesting illustrations.

Perhaps the major criticism of the book is that the author has used secondary sources almost exclusively: of 122 bibliographic references, only 22 date to before 1900. The author, however, is scrupulous in recognizing his authorities.—[*History of Veterinary Medicine*. By Valentino Chiodi. 535 pages; 177 illustrations, 8 in color. Farmitalia, Milano, Italy. 1957. Price not given.]—J. F. SMITHCORS.

THE NEWS

Tours Planned for XVIth International Veterinary Congress in Madrid in May, 1959

The U.S. Committee for the XVth International Veterinary Congress to be held in Madrid, Spain, May 21-27, 1959 (see the JOURNAL, June 1, Page 500), has completed arrangements with the Travel Service Bureau, Inc., Needham, Mass., to offer a selection of group travel plans, including travel to Madrid and post-Congress tours to other points of interest in Europe.

A preliminary tour leaflet, describing the four different travel plans to be offered, was mailed early in September to AVMA members in the United States, Canada, and the West Indies. Travel to Madrid can be made by steamship or by air; return to the States will offer the same choice of transportation. The duration of the four tours is from one week (for those who want to attend only the Congress in Madrid) to two, three, and four weeks. This "a la carte" choice also applies to the itineraries offered. The preliminary tour leaflet contains a return form so that interested persons can obtain complete information regarding itineraries, costs, etc.

Travel Service Bureau is the agency which handled the group travel arrangements so satisfactorily for U.S. participants in the XVth I.V.C. in Stockholm, Sweden, in 1953. Over 100 persons joined in the tours to England, Scotland, Norway, Sweden, and other points on the Continent.

Anyone who failed to receive the preliminary announcement and tour leaflet can obtain one from the AVMA office, 600 S. Michigan Ave., Chicago 5, Ill.; or by writing to Travel Service Bureau, 32 Dedham Ave., Needham, Mass., attention Mr. N. M. Jost.

Additional information about the scientific program, entertainment, and social features of the Madrid Congress will be published in the JOURNAL from time to time as received from the Organizing Committee in Spain.

Veterinary Anatomical Nomenclature

The American Association of Veterinary Anatomists, organized in 1949, each year sponsors a standing committee on nomenclature. Its members have kept in close touch with a similar committee affiliated with the American Association of Anatomists which drafted the Parisian nomenclature.

With the retirement of Dr. Grossman and Dr. Foust, a revised committee was appointed as follows: Drs. R. Getty, Iowa State College; R. Kitchell, University of Minnesota; M. E. Miller, New York State Veterinary College; D. M. Trotter, Kansas State College; L. E.

St. Clair, University of Illinois; R. W. Davis, Colorado State University; J. A. McCurdy, Washington State College.

This committee held a two-day meeting at Columbus, Ohio, Aug. 14-15, 1957. At this meeting, Dr. Ralph Kitchell was appointed to represent the American veterinary anatomists at the International Veterinary Nomenclature Committee meeting held in Freiburg, Germany, in September, 1957. This appointment was given official sanction by the AVMA through Dr. Hardenbergh, executive secretary of that Association.

While circumstances prevented Dr. Kitchell from attending the meeting, he has since held conferences with Dr. Josef Schreiber, chairman of the Nomenclature Commission of the International Association of Veterinary Anatomists. In September, 1957, a request was sent from the A.A.V.A. to Dr. Schreiber, in Vienna, Austria, petitioning for active membership in the international association.

During the past year this committee, with its various subcommittees, has been working toward a revision of the "Nomina Anatomica Veterinaria" (1923) to coincide with the Paris revision of the "Nomina Anatomica" (1956). In the meantime, Dr. Kitchell visited many of the veterinary anatomists abroad and discussed the problems of nomenclature with them. The commission will be truly international in scope and the future looks very promising.

A meeting of the International Association of Veterinary Anatomists will be held in Copenhagen, October, 1958. The subject of nomenclature will form an important part of the agenda. The A.A.V.A. hopes that one of its members, or another suitable representative, will be able to attend the conference as delegate. Anyone interested in such an assignment may contact Dr. L. E. St. Clair, Anatomy Department, University of Illinois, Urbana, Ill.

A veterinary appendix to the Paris nomenclature, or some other appropriate revision, is expected to be ready for the International Veterinary Congress in Madrid, in 1959.

AMONG THE STATES AND PROVINCES

District of Columbia

District of Columbia Academy of Veterinary Medicine.—Dr. Jean Holzworth of the Angell Memorial Animal Hospital, Boston, Mass., was the guest speaker at the June 3, 1958, meeting of the academy.

During the course of the two-hour presentation, Dr. Holzworth discussed and illustrated by koda-chrome projections several important feline disease entities. The main portion of her paper was devoted to feline infectious anemia and blood dyscrasia.

Emphasis was placed upon the need for blood examination by practitioners in order to properly evaluate and correlate clinical syndromes for more accurate differential diagnosis. Skin disease and some new approaches to its treatment, iliac thrombosis, and the clinical symptoms associated with this syndrome, and feline toxoplasmosis were also explored. A brief question and answer period followed.

s/A. BUDD FENTON, *Corresponding Secretary.*

Iowa

Annual Conference for Veterinarians.—Iowa's annual conference for veterinarians was conducted at Iowa State College, Division of Veterinary Medicine, Ames, July 22-23, 1958.

Dr. James H. Hilton, president, welcomed the conferees at the afternoon session, July 22, at which Dean I. A. Merchant presided. Drs. M. W. Swenson and M. A. Emmerson, both of Ames, presided at the remaining sessions of the conference. Dr. R. A. Packer, Ames, was program chairman. The principal speaker at the centennial banquet held in the Memorial Union, July 22, was Dr. Margaret W. Sloss, Department of Veterinary Pathology at Iowa State.

Among the several TV demonstrations that were presented included: Drs. M. A. Emmerson—X Ray Hazards; D. L. Baker—Dental Care of the Canine Patient; R. A. Packer—Mastitis Diagnosis, Field, and Laboratory Tests; B. W. Kingrey and R. L. Lundvall—Immobilization of Fractures of Large Animals and Anesthetic Technique for Large Animals.

In the field of large animals, Dr. E. A. Carbreys discussed the "Incidence of Bovine Leptospirosis in Iowa" and Dr. R. L. Lundvall reported on "Shetland Pony Practice."

With regard to small animals, Dr. J. Bailey talked on "Scabies in Sheep;" Drs. S. H. McNutt spoke on the "Current Outlook for Control of Hog Cholera" and J. E. Lovell delivered an address on "Artificial Insemination in Swine."

Discussions of a general nature included: Drs. P. T. Pearson—Handling the Nephritis Patient; John B. Herrick—Mastitis Control Program; Earle Raun—Systemic Insecticides; and Dean I. A. Merchant—Research in Veterinary Medicine at Iowa State College.

During the last session of the conference a movie entitled "Gamma Radiation Equipment and Its Use on Animal Pathogens" was presented by Dr. J. C. Picken of the Veterinary Medical Research Institute.

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Central Iowa Association.—The Central Iowa V.M.A. met at the Breese House in Ankeny on June 16, 1958.

A panel discussion was held on the problems encountered in swine practice. Dr. D. V. Dugan, Minburn, Iowa, moderated the conference.

s/S. L. HENDRICKS, *Secretary-Treasurer.*

Nine Veterinarians Honored by ISC Alumni Association.—Nine veterinarians were among the men and women who were honored by the Iowa State College Alumni Association at a centennial recognition luncheon held on campus in the Memorial Union, June 14, 1958. This luncheon is part of a series of events being presented during the year in observance of the one hundredth anniversary of the founding of Iowa State College.

Seven of the veterinarians were among the 100 who received a centennial citation for having "made a unique contribution to the stature of Iowa State College." They are: Drs. Burton J. Gray (ISC '43), president of Fort Dodge Laboratories, Inc.; Aaron H. Groth, Sr. (ISC '21), dean of the School of Veterinary Medicine, University of Missouri; Earl A. Hewitt (ISC '18), professor of physiology and pharmacology, Division of Veterinary Medicine, Iowa State College; Charles Murray (ISC '10), dean emeritus, Division of Veterinary Medicine, Iowa State College; Harry L. Foust (OSU '14), technical advisor to Pakistan, Dacca, East Pakistan; M. Lois Calhoun (ISC '39), professor and head, Department of Veterinary Anatomy, College of Veterinary Medicine, Michigan State University; and Carl Olson, Jr. (ISC '31), professor of veterinary science, University of Wisconsin.

An Iowa State College Alumni Merit Award was presented to Dr. Samuel H. McNutt (ISC '17), head of the Department of Veterinary Science, University of Wisconsin, "in recognition of preeminent service in advancing human welfare."

Dr. Ival A. Merchant, dean of the veterinary school, received a faculty citation for his long and outstanding service on the college's staff.

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Cedar Valley Association.—The officers elected at the June 9, 1958, meeting of the Cedar Valley V.M.A. were as follows: Drs. J. S. Guldner, Waterloo, president; R. A. Benson, Dunkerton, vice-president; and S. L. Diesch, Winthrop, secretary-treasurer.

s/S. L. DIESCH, *Secretary-Treasurer.*

Kansas

Veterinary Association and Foundation Established.—During the twentieth annual conference for Kansas veterinarians held June 5-7, 1958, in Manhattan, the veterinary alumni of Kansas State College formed the Kansas State College Veterinary Medical Alumni Association.

One of the first acts of the new association was to establish the Kansas State College Foundation for Veterinary Medicine. This foundation will be administered by the Kansas State College Endowment Association. The funds from the foundation may be used for

graduate and undergraduate scholarships, for short term grants to veterinary school staff members for postgraduate training, and for special research projects and other activities for which finances are not available from other sources. The foundation now estimates its fund to be approximately \$1,000.

The officers elected to serve the alumni association for the coming year are as follows: Drs. J. F. Knappenberger, president; W. O. Kester, president-elect; Fayne H. Oberst, secretary-treasurer; and R. L. Anderes, two-year term, and Fred Walker one year term, both of whom are members-at-large.

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Kansas State College's Annual Awards Banquet.—During the banquet held at the Wareham Hotel on May 24, 1958, Dean E. E. Leasure announced the numerous prizes and certificates of merit that were awarded to some of the students in the School of Veterinary Medicine. They are as follows:

Women's Auxiliary to the American Veterinary Medical Association (\$50) for outstanding leadership among students: James D. Smith.

Kansas V.M.A. Awards and Certificates for General Proficiency in the four years of the professional curriculum: Wallace B. Wren (\$15) and Norman Max Held (\$10)

Kansas V.M.A. Awards and Certificates to students most proficient in large and small animal clinics: Keith B. Beeman (\$15), large animal; Donald L. F. Pohlman (\$15), small animal.

Merck Veterinary Manual Award to two seniors for outstanding achievement of a special nature in veterinary medicine: Farrel Robinson and Gerald D. Rousseau.

Phi Kappa Phi Membership for highest scholarship: Kenneth D. Weide, Max L. Sutton, Wallace B. Wren, Robert F. Sand, and George W. Olson.

Gamma Sigma Delta Scholarship Society in Agriculture and Related Sciences awarded membership to seven members of the senior class: George W. Olson, James H. Sherrod, Hal R. Sinclair, Nickolas J. Sojka, Max L. Sutton, Kenneth D. Weide, and Wallace B. Wren.

The Borden Award (\$300) for high scholarship during the first six semesters of the professional curriculum in veterinary medicine: (\$300): Kenneth D. Weide.

The O. M. Franklin ('12) Award (\$100) for scholarship and need: Robert F. Sand.—*Veterinary Alumni News Kansas State College, School of Veterinary Medicine* (July 1, 1958): 38.

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Kansas City Association.—The Kansas City V.M.A. held its regular meeting on July 17, 1958, in the Aztec Room of the Hotel President.

The speakers for the evening were Dr. W. Wolfe and Dr. N. B. Tennille, both of whom are instructors in the School of Veterinary Medicine at Oklahoma State University. Dr. Wolfe spoke on "Selected Case Reports in Large Animal Practice" and Dr. Tennille discussed "The Treatment of Emergency Cases in Small Animal Practice."

S/FRANK A. O'DONNELL, Secretary.

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Dr. Borgman Working on a Postdoctoral Research Fellowship.—Dr. R. F. Borgman (MSC '47), Manhattan, has begun work on a postdoctoral research fellowship from the National Institute of Arthritis and Metabolic Diseases, National Institutes of Health.

He plans to undertake studies of nutritional muscular dystrophy at the physiology department at Kansas State College. The period of the grant is for one year.

Michigan

Nationwide Tuberculosis Eradication Conference Held at Michigan State University.—

A national tuberculosis eradication conference was held June 15-20, 1958, on Michigan State University's campus.

Sponsored jointly by the Animal Disease Eradication Division of the Agricultural Research Division, U.S.D.A., and the College of Veterinary Medicine at Michigan State, the conference was planned primarily for the training of federal veterinarians who will lead the final "push" for the eradication of tuberculosis from the continental United States and Puerto Rico.

The conference was attended by 49 U.S.D.A. veterinarians, representatives from about half of the veterinary colleges, and many distinguished speakers and guests. The five-day conference covered every phase of the history, diagnosis, prevention, and control of tuberculosis, including practice testing clinics and instruction in necropsy techniques. Both "live" specimens and closed-circuit television demonstrations were used.

A high point of the conference was the presentation, at the banquet on June 19, by the ARS of a Certificate of Appreciation to Dr. Howard R. Smith, formerly general manager of the National Livestock Loss Prevention Board and a pioneer in the early promotion of laws for the control of tuberculosis in livestock.

S/W. W. ARMISTEAD, Dean, College of Veterinary Medicine, Michigan State University.

Minnesota

Dr. Donald to Be a Consultant in Surgical Research at the Mayo Clinic.—Dr. David E. Donald, a graduate of the Royal (Dick) Veterinary College at the University of Edinburgh

in 1943, has been appointed a consultant in surgical research at the Mayo Clinic in Rochester, Minn.

Dr. Donald entered the Mayo Foundation in April, 1950, as a fellow in experimental surgery, and later transferred to physiology. He had formerly served as a research assistant, research associate, and as an assistant to the staff at the Mayo Clinic.

In June, 1958, he received a Ph.D. degree in physiology from the University of Minnesota.

New Jersey

State Certified as Modified Brucellosis-Free Area.—On June 17, 1958, U.S. Secretary of Agriculture signed the certificate that gave New Jersey its long-sought official rating of "modified brucellosis-free."

To celebrate the achievement of this goal, reached after 32 years of joint efforts of livestock disease control officials, dairymen, public health officers and others, the State Board of Agriculture sponsored a "brucellosis certification luncheon" at Far Hills on July 1, which was attended by representatives of the various groups.

The state's brucellosis control program was begun in 1926 at which time more than 20 per cent of the cattle tested were found to be infected. The reactor rate is now well below the 1 per cent limit for certification as a modified brucellosis-free area.—*Farm Service News, N.J. Dept. of Agriculture, July, 1958.*

North Carolina

State Association.—The fifty-seventh annual meeting of the North Carolina State V.M.A. met at the Washington Duke Hotel in Durham, N. Car., June 24-26, 1958. Over 100 veterinarians registered. Dr. C. H. Gurley was chairman of the committee on local arrangements.

The preconvention session, held at the Washington Duke Hotel on June 24, was presided over by Drs. D. C. Beard, Concord, W. R. Dobbs, Albermarle, C. R. Swearingen, Smithfield, and B. H. Kinsey, Washington.

Beside the preconvention session, demonstrations were also presented at three of the other sessions of the conference. Among the demonstrations were: Drs. W. E. Plummer—Restraint for Castration; R. W. Smith—Reduction of Fractured Radius; W. D. Collins—Corneal Transplants; and M. J. Tillery—Scrapie.

On June 25, a question and answer luncheon was presided over by Dr. W. O. Slappey, Fayetteville. Dr. A. W. Smith, Farmville, moderated a panel discussion on the "Control of Infectious Diseases" the first day of the meeting and Dr. M. P. Hines mediated another panel on the same topic the second day of the conference.

One of the highlights of the fifty-seventh annual banquet held June 25 was the presentation of the "Veterinarian of the Year" Award to Dr. J. I. Neal of Southern Pines.

The roster of officers for 1958-1959 was also announced at the banquet. The following veterinarians will serve for the ensuing year: Drs. B. H. Kinsey, Washington, president; C. R. Swearingen, Smithfield, president-elect; C. J. Lange, Greensboro, vice-president; J. T. Dixon, Winston-Salem, secretary-treasurer.

Dr. A. A. Husman is delegate and Dr. M. P. Hines is alternate to the AVMA Convention in Philadelphia. Both veterinarians are from Raleigh, N. Car.

Members of the executive board of the North Carolina State V.M.A. are: Drs. A. W. Smith, Farmville; W. R. Dobbs, Albermarle; C. C. McLean, Southern Pines; D. C. Beard, Concord; and R. T. Stapleton, Fayetteville.

In 1959, the fifty-eighth annual meeting of the association will be held June 24-25, at the Morehead Biltmore, Morehead City, N. Car.

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Dr. Neal Selected as "Veterinarian of the Year" by the North Carolina State V.M.A.—Dr. Julius Issac Neal (KVC '15), Southern Pines, was chosen as the North Carolina State Veterinary Medical Association's "Veterinarian of the Year" on June 25, 1958.



Dr. J. I. Neal (right), named "Veterinarian of the Year" by the North Carolina V.M.A., is congratulated by Dr. D. C. Beard, president, at the association's fifty-seventh annual meeting held in Durham on June 25, 1958.

Dr. Neal was cited for his "outstanding and meritorious service to the livestock industry and veterinary profession of the state" and presented with an inscribed plaque by Dr. D. C. Beard, Concord, president, at the annual banquet held at the Washington Drake Hotel.

Dr. Neal is widely known for his ability in the diagnosing and treatment of lameness in horses. During the course of his practice, many of his clients have had horses win fame. Among these have been Millbrook, Chuck Volo, Newport Dream, Bright Lights, Times Square, King Commander, and Rythemimhim.

Dr. Neal began his practice in Sanford, N. Car., in 1915. His practice called him to Southern Pines and Pinehurst several time each week and he soon established an office at the race track in Pinehurst.

Active in the North Carolina State V.M.A., Dr. Neal has served as president of the organization and also as chairman of the board of veterinary medical examiners. He is a member of the AVMA, the American Animal Hospital Association, the Equine Practitioners Association, and the Kiwanis Club.

Quebec

Saint-Hyacinthe Again Sponsors Agricultural Products Course.—For the eighth consecutive year, special courses on the preparation and the marketing of agricultural products have been given recently at the School of Veterinary Medicine, Saint Hyacinthe.

These courses have been made possible due to the cooperation of the Division of Agriculture of the provincial and federal departments of agriculture in collaboration with the province's Department of Social Welfare and Youth and with the Federal Department of (Public) Work.

This series of courses has proved to be very valuable to the agriculturist particularly to the agriculturist in the Province of Quebec.

s/DR. JACQUES SAINT-GEORGES, Secretary.

COMMENCEMENTS

Graduating Class, 1958, School of Veterinary Medicine, Tuskegee Institute



Top row (left to right)—Albert Carey, James Bryant, Paul Allen, Thelma Dean, Emanuel Butler, Kermit Cockrell, Russell Draw.

Second row—David Geiger, Robert Turnquest.

Third row—Alfred Godwin, Pedro Sastre-Maysonet, T. S. Williams, dean; Carlos Stella-Cintron, Harold Turnquest.

Fourth row—Edward Robinson, Dean R. Hodges, Theodore Jamison, Robert Johnson, Harold Lape-sarde, Adam McKee, Jr., Curtis Williams.

Tuskegee Institute.—At the 1958 commencement exercises of the School of Veterinary Medicine, Tuskegee, Alabama, the following 20 candidates were presented for the D.V.M. degree:

Paul Allen
James Bryant
Emanuel Butler
Albert Carey
Kermit Cockrell
Thelma Dean
Russell Drew
David Geiger
Alfred Godwin
Dean R. Hodges

Theodore Jamison
Robert Johnson
Harold Lapesarde
Adam McKee, Jr.
Edward Robinson
Pedro Sotomayor
Carlos Stella-Cintrón
Harold Turnquest
Robert Turnquest
Curtis Williams

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Ohio State University.—At the 1958 commencement exercises of the College of Veterinary Medicine, Ohio State University, the following 67 candidates were presented for the D.V.M. degree:

Robert G. Armstrong
John C. Barck
Donald R. Bartels
Charles W. Beall
Roland D. Berlekamp
John F. Bowers
Frederick E. Boyer
Homer Brickman
William Ben Bright
Lowell Ned Brown
Ralph Burkholder

Richard E. Burwell
William M. Busey
Harold L. Butcher
Joseph T. Cain
David E. Caldwell
Joseph T. Closson
Warren R. Compton
Thomas L. Connair
Robert Costigan
Edgar Craig DeLong
James R. Droeze

Graduating Class, 1958, College of Veterinary Medicine, Ohio State University



Top row (left to right)—L. Meredith, A. Endo, D. Kerns, P. Mancinotti, N. Brown, B. Bright.
Second row—W. Kuhlmann, R. Berlekamp, R. Weadick, D. Zuck, L. Pond, R. Woodgeard.
Third row—J. Houdeshell, J. Stump, S. Richeson, A. George, D. Bartels, J. Huber, J. Droeze, A. Graff, F. Boyer, D. Nofsinger, T. Wittich.
Fourth row—G. Headley, W. MacKenzie, J. Herman, R. Burkholder, R. Armstrong, R. Nash, T. Connair, H. Hartman, J. Wood, J. Noonan, R. Costigan.
Fifth row—B. Epperson, P. Linerode, F. Goldstein, J. Cain, J. Barck, D. Yundt, W. Busey, R. Hamlin, D. Recker, F. Joos, J. Rychener.
Sixth row—D. Caldwell, J. Eppey, B. Griffith, H. Brickman, C. Miller, D. Johnson, H. Butcher, C. Hale, J. Quecke, R. Gruesser, J. Vickers.
Seventh row—R. Burwell, W. Compton, J. Closson, C. Beall, R. Fuller, L. Maxwell, J. Bowers, R. Glaab, W. Palta, C. DeLong, C. Purdy.

Arthur K. Endo
Boyd Epperson
John R. Eppley
Ronald R. Fuller
Alan E. George
Robert A. Glaab
Fred G. Goldstein
Alverda D. Graff
Bob G. Griffith
Roger J. Gruesser
Charles H. Hale
Robert L. Hamlin
Howard A. Hartman
Maurice G. Headley
James E. Herman
Jesse W. Houdeshell
Jan D. Huber
Robert R. Johnson
Francis E. Joos
Donald G. Kerns
William Kuhlmann
Phillip A. Linerode

William F. MacKenzie
Paul Mancinotti
Lewis Maxwell
Leavitt Meredith
Cecil R. Miller
Rex A. Nash
Donald C. Nofzinger
John P. Noonan
William J. Palte
Leland A. Pond
Clyde L. Purdy
Justin J. Quecke
Daniel L. Recker
Samuel S. Richeson
John Rychener
John E. Stump
James H. Vickers
Robert J. Weadick
Thomas A. Wittich
John B. Wood
Robert L. Woodgeard
David H. Yundt

David R. Zuck

University of Pennsylvania.—At the 1958 commencement exercises of the School of Veterinary Medicine, University of Pennsylvania, the following 46 candidates were presented for the D.V.M. degree:

Ignatius W. Adams
Wiley V. Behler
George Bjorvold
Richard L. Carp
Richard L. Cook
L. Robert Davenport
Robert W. Despres
Sheldon S. Diamond
George F. Durfee
Peter P. Fenchak
George L. Flickinger
Daniel V. Flynn
Allen W. Hayen
Ronald T. Hopwood
Donald W. Hostettler
Bernard S. Jortner
Natalie L. Knowles
George W. Leighow
Gerald E. Lewis
Morten S. Mael
James S. McFarland
Gaylord E. McKissick
Carlos E. Montes

Lester D. Morris
George F. Orthey
Robert W. Pfeiffer
Robert L. Ricker
Lionel Rubin
Frederick R. Rude
John B. Schmidt
Mead F. Shaffer, Jr.
Edward T. Siegel
Donald A. Skinner
Russell J. Snyder
Richard H. Stoneback
B. Joseph Tibolla
Glen C. Todd
Stanley O. Travis
Donald G. Vebrust
Shirley R. Walters
Beecher H. Watson
William R. Williams
George H. Wyckoff, Jr.
Paul A. Wysock
V. William Zeman
Curtis A. Zillhardt

FOREIGN NEWS

Colombia

Colegio Medico Veterinario.—Since January, 1958, the Colegio Medico Veterinario del Valle del Cauca has been holding monthly meetings in the different towns of the Valle del Cauca Department.

The monthly meetings of the college in 1958 were scheduled as follows: January 14, February 15, March 15, April 19, May 17, June 21, July 12, with the last meeting held August 15-17 at Armenia.

The Colegio Medico Veterinario was organized at Cali, the main city in the southwestern portion of Colombia, South America, which has a population of 400,000.

s/JOSE GONZALEZ ALVAREZ, Correspondent.

STATE BOARD EXAMINATIONS

Interested persons can obtain information about applications, fees, deadlines for filing applications, and exact time and place of examinations of the respective boards by writing to the persons whose names and addresses are given below.

BRITISH COLUMBIA—November, 1958 (usually end of the second week; Vancouver. G. L. Stovell, British Columbia Veterinary Association, 3187 West 43rd Ave., Vancouver 13, secretary.

U.S. GOVERNMENT

Veterinary Personnel Changes.—The following changes in the force of veterinarians in the U.S.D.A. are reported as of June and July 2, 8, 9, and 11, 1958.

TRANSFERS

Leo G. Berg, from Springfield, Ill., to Harrisburg, Pa.
S. J. Berger, from Indianapolis, Ind., to Boston, Mass.
Meier Brodner, from New York, N. Y., to Washington, D. C.
William O. Caplinger, from Philadelphia, Pa., to Washington, D. C.
Hunter Cohen, from Newark, N. J., to Buffalo, N. Y.
Meyer W. Cohen, from Oklahoma City, Okla., to Philadelphia, Pa.
Laval N. Cothran, from Greenport, L. I., N. Y., to Philadelphia, Pa.
W. L. Downey, from Denver, Colo., to Montpelier, Vt.
Arthur F. Eckert, from Los Angeles, Calif., to Washington, D. C.
James A. Faughn, from St. Louis, Mo., to Union City, Tenn.
Charles A. Franz, from St. Louis, Mo., to Washington, D. C.
Dale W. Glascock, from Washington, D. C., to Chicago, Ill.
Lawrence F. Good, from Jefferson City, Mo., to Salt Lake City, Utah.
Garner J. Grissam, from San Antonio, Texas, to Gaffney, S. Car.
Clare C. Hamilton, from Norfolk, Va., to Davenport, Iowa.
Claude D. Hinkley, from Milan, Ill., to Cedar Rapids, Iowa.
Herndon P. Honestead, from Springfield, Ill., to Richmond, Va.
William S. Houk, from Denver, Colo., to St. Joseph, Mo.
M. B. Huffman, from Frankfort, Ky., to Baton Rouge, La.
Frank R. Huster, Jr., from St. Louis, Mo., to Fort Worth, Texas.
Gordon C. Janney, from Madison, Wis., to Beltsville, Md.
Irving J. Kahn, from New York, N. Y., to Los Angeles, Calif.
David H. Kelley, from Nashville, Tenn., to Jackson, Miss.
Robert F. Kielsen, from Fort Worth, Texas, to Milwaukee, Wis.
Abby J. Logie, from Philadelphia, Pa., to Norfolk, Va.
Harry B. Mitchell, from Waterloo, Iowa, to Green Bay, Wis.
Thomas A. Moir, from Milwaukee, Wis., to St. Louis, Mo.
Charles E. Mootz, from Chicago, Ill., to Detroit, Mich.
William M. Moulton, from Richmond, Va., to Albany, N. Y.
S. K. Nelson, from Boston, Mass., to New York, N. Y.

Melvin O. Nottingham, from Albuquerque, N. M., to Indianapolis, Ind.
Thomas M. Nunley, from San Antonio, Texas, to Birmingham, Ala.
Otis C. Post, from Jacksonville, Fla., to Fort Worth, Texas.
Clayton J. Price, from Scottsbluff, Neb., to Oklahoma City, Okla.
Tihomir Rad, from Milwaukee, Wis., to Waterloo, Iowa.
Nicola Romano, from Chicago, Ill., to Los Angeles, Calif.
Otto W. Seher, from Chicago, Ill., to Washington, D.C.
Harry S. Shanklin, Jr., from Richmond, Va., to Montgomery, Ala.
George C. Stewart, Jr., from Nashville, Tenn., to Phoenix, Ariz.
James H. Stewart, from Worthington, Minn., to Fremont, Neb.
Philip Telishevsky, from Baltimore, Md., to Cleveland, Ohio.
Arthur R. Thiele, from New York, N.Y., to Newark, N. J.
W. T. Vaught, from Richmond, Va., to Ames, Iowa.
William J. Waters, Jr., from Gaffney, S. Car., to St. Louis, Mo.
Donald R. Wenger, from Worthington, Minn., to Tecumseh, Neb.
Adelbert E. Wilcox, from Tecumseh, Neb., to Worthington, Minn.

RETIREMENTS

Oscar W. Anderson, Davenport, Iowa.
Roy Avnat, Nashville, Tenn.
Marion L. Cravens, Detroit, Mich.
Frank P. Miller, Kansas City, Kan.
Homer S. Perdue, Cedar Rapids, Iowa.
John H. Winter, Columbus, Ohio.

DEATHS

Star Indicates Member of AVMA

Harry C. Barth (CVC '05), 76, Amboy, Ill., long-time Lee County veterinarian, died June 9, 1958, at Glenview where he had been living with a daughter, Mrs. Frederick Priess.

Dr. Barth had practiced in Amboy and in Lee County for more than 50 years before retiring. Surviving, beside his daughter, are two brothers and three sisters.

★William Luther Coleman (CVC '19), 76, Albany, Ill., died suddenly June 22, 1958, at his home.

Born in Albany Township, Dr. Coleman received his early education in the Newton School. After graduation from the Chicago Veterinary College, he began his practice in the Albany area. Dr. and Mrs. Coleman have made their home in Albany for the last 19 years.

Dr. Coleman was a member of the Illinois State V.M.A., the Albany O.E.S. Moline Consistory, and the Presbyterian Church. He was also a 35 year member of the Albany Masonic Lodge.

Surviving are his widow, Jessie M. Kelly Coleman, a brother, a niece, and a nephew.

★Benjamin F. Corbin (OSU '35), 59, Culver City, Calif., died June 1, 1958, at his home.

Dr. Corbin operated the Culver City Cat and

Dog Hospital for many years. He was a member of the Florence Masonic Lodge 649, Scottish Rite, Al Malaikah Shrine Temple, and the Culver City Moose Lodge. He was also a past-president of the Culver City Lions Club, serving from 1949 to 1950, a charter member of the Culver City Elks Lodge 1917, Order of the Eastern Star, and the American Legion Community Post 46.

He is survived by his widow, three daughters, and a son.

Howard A. Downey (KCV '18), 67, Appleton, Wis., died June 8, 1958, after an illness of one week.

Dr. Downey had lived in Appleton for the last 33 years. Before retiring, he had been associated with the Bureau of Animal Industry in the tuberculosis eradication section.

He is survived by his widow and one son, Dr. Keith W. (KSC '42), of Green Bay; three brothers, and two sisters.

R. J. Hight (CVC '07), 73, Phoenix, Ariz., died in his home after a lengthy illness.

Born in Vienna, Ill., Dr. Hight had resided in Tempe, Ariz., for 44 years before moving to Phoenix a year ago. In Tempe, Dr. Hight engaged in farming, dairying, and in cattle raising and feeding. He had served on the city council and was a past-president of the Salt River Valley Water Users Association. He had also been a long-time member and a past-president of the Rotary Club there.

Dr. Hight was a member of the Arizona Cattle Growers Association, the Arizona Club, and had served on the Roosevelt Council of the Boy Scouts of America. At various times, he also served as state veterinarian and, in 1950, Dr. Hight was the Republican candidate for secretary of state.

Surviving him are his widow, Hollie Clymer Hight, a daughter, and three grandchildren.

Sidney Holmes (MCK '14), 77, Marengo, Ill., who has resided for the past 12 years with Mr. and Mrs. Steve Otis, died June 16, 1958.

Dr. Holmes was born in England in 1881 and attended a veterinary college there. He served with the English Army during World War I and came to America at the end of the war, graduating from the now extinct McKillip Veterinary College in Chicago.

Dr. Holmes had lived in Marengo since 1930 and served as state veterinarian for 25 years, retiring in 1957. He is survived by a niece and two nephews.

★William R. Jackson (KSC '46), 34, Rogers, Ark., a member of the city council and fire department volunteers, died at the Rogers Memorial Hospital, June 30, 1958, following an extended illness.

Dr. Jackson was born at Neosho, Mo., and attended elementary and high school there. He established his practice in Rogers soon after his graduation from Kansas State College and operated an office and a kennel in Rogers for 12 years, until illness forced his retirement a year ago.

His widow, Geneva, three daughters, and a sister survive Dr. Jackson.

Alexander Junkunc, Sr., 82, Mt. Prospect, Ill., retired cofounder of the J. & J. Tool & Machine Company, died in his home June 12, 1958.

Before leaving Hungary in 1923, Dr. Junkunc was veterinary surgeon of the Hungarian government. He is survived by his widow, Mrs. Martha Junkunc, four sons, and a daughter.

Fred Main (GR '08), 82, Albion, Mich., died June 10, 1958, at McPherson Health Center Hospital, Howell, where he had been a patient for three weeks.

Dr. Main had practiced in Albion for about 40 years prior to his retirement in 1947. He was an Albion alderman in 1915 and was county veterinarian from 1934 to 1936. Dr. Main also served several terms as city milk inspector.

He is survived by his widow, Mame Main, and three sons.

E. C. McConnell (IND '10), 89, Cynthiana, Ind., died May 21, 1958. He had been in ill health and confined to his bed for the past three months.

Dr. McConnell retired from his practice about 15 years ago. He is survived by three daughters.

Charles E. Price (SF '06), 81, Santa Ana, Calif., died at his home, June 12, 1958, after a prolonged illness.

Dr. Price was a lifetime resident of Orange County. He retired in 1956, after 50 years of practice. He was a member of the Native Sons of the Golden West and the Izaak Walton League. His widow, two sons, and two sisters survive Dr. Price.

***Ralph N. Seamon** (GA '56), 26, Johnson City, Tenn., died June 24, 1958, in a Lexington, Ky., hospital of injuries received in an automobile crash there June 19.

Dr. Seamon attended Emory-at-Oxford and subsequently graduated from the University of Georgia in 1956.

Surviving Dr. Seamon are his widow, Vesta Dobbs Seamon, two daughters, and a son.

***Lloyd Banks Sholl** (COR '23), 64, Okemos, Mich., died in a Lansing, Mich., hospital after a brief illness on July 14, 1958. Dr. Sholl professor of veterinary pathology at Michigan

State University at the time of his illness, was to have retired in June, 1959, after 35 years of service to the university.

A long-time member of the AVMA, Dr. Sholl was a frequent contributor to the veterinary literature in the field of clinical pathology. Other affiliations included the Michigan V.M.A., American Association of Pathologists, Phi Zeta, Alpha Psi, Sigma Xi, and Phi Kappa Phi.

A veteran of World War I, Dr. Sholl was a past-commander of the East Lansing Post of the American Legion as well as a member of the Okemos Masonic Lodge. He is survived by his widow, Helen, and three sisters. Burial was in Arlington Cemetery in Milwaukee, Wis.

Lee C. Smith (KCV '08), 75, Ventura, Calif., died suddenly of a heart ailment on June 9, 1958.

Born on a farm near Tucker's Ford, northwest of Carthage, Mo., Dr. Smith began his practice in Hamilton, Mo., where he practiced until entering the service of the Bureau of Animal Industry about 25 years ago; he was an inspector in Missouri, Arkansas, Texas, and California. He retired ten years ago.

Surviving Dr. Smith are his widow, Susan Giddings Smith, four sisters, and a brother.

Walter Ervin Stone, Sr., (MCK '16), 77, Liberty, Mo., died May 31, 1958, in a St. Joseph, Mo., hospital.

Dr. Stone had practiced in the Liberty area for 32 years before ill health forced his retirement two years ago. Surviving Dr. Stone are his widow, Bessie L. Stone, a son, and a brother.

***Fritz Volkmar** (OSU '31), 75, Downey, Calif., died April 4, 1958.

Dr. Volkmar received his preliminary education at the University of Munich and the University of Giessen. After obtaining his D.V.M. degree in 1931, Dr. Volkmar continued on at Ohio State University where he obtained his M.S. in 1932.

A member of the Sigma Xi and Phi Zeta, Dr. Volkmar was also affiliated with Reichsverband der Deutschen Tierärzte and the Chicago V.M.A.

***Raleigh P. Wiese** (CVC '17), 62, Garretson, S. Dak., a retired colonel in the U.S. Army, died suddenly at his home, June 17, 1958, of a heart attack.

Born in Mt. Joy, Iowa, Dr. Wiese established his residence in Garretson in 1917, after his graduation from the Chicago Veterinary College. He served in the Army for 31 years.

Dr. Wiese is survived by his widow, Ella M. Erickson Wiese, a daughter, and a son.

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R. G. Schirmer, D.V.M., M.S.,
F. E. Ends, D.V.M., M.S., and
J. P. Newman, D.V.M., M.S.

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by R. W. Barnes, D.V.M.

Reprint, The North American
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by S. Lester Jackson, D.V.M.

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Blood Level Studies in Dogs Following the Administration of Chloromycetin

F. E. Ends, D.V.M., M.S.,
A. J. Glazko, Ph.D.,
L. M. Wolf, B.S.,
John Ehrlich, Ph.D.,
M. Galbraith, M.S.

BLOOD LEVEL STUDIES IN DOGS FOLLOWING THE ADMINISTRATION OF CHLOROMYCETIN

by F. E. Ends, D.V.M., M.S.,
A. J. Glazko, Ph.D.,
L. M. Wolf, B.S.,
John Ehrlich, Ph.D.,
M. Galbraith, M.S.

Reprint from the American
Journal of Veterinary
Research

The blood levels of chloromycetin in dogs following the administration of chloromycetin are shown in the following table:

Time (hours)	Blood Level (mg/100 ml)
0	0
1	1.2
2	1.5
4	1.8
6	1.6
8	1.4
10	1.2
12	1.0
14	0.8
16	0.6
18	0.4
20	0.2
22	0.1
24	0.0

THE PRACTICAL RESULTS OF SENSITIVITY TESTS IN SMALL ANIMAL PRACTICE

by Margaret Schlichting, S.A.

Reprint from Veterinary Medicine

BOVINE BLOOD SERUM CONCENTRATIONS OF CHLOROMYCETIN FOLLOWING INTRAMUSCULAR ADMINISTRATION

by F. E. Ends, D.V.M., M.S.
and K. D. Van Nocker,
D.V.M.

The bovine blood serum concentrations of chloromycetin following intramuscular administration are shown in the following table:

Time (hours)	Blood Serum Concentration (mg/100 ml)
0	0
1	1.2
2	1.5
4	1.8
6	1.6
8	1.4
10	1.2
12	1.0
14	0.8
16	0.6
18	0.4
20	0.2
22	0.1
24	0.0

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In accordance with paragraph (e) of Section 2, Article X, of the Administrative Bylaws, the names of applicants residing within the jurisdictional limits of a constituent association shall be published once in the JOURNAL.

The following applicants have been certified by the secretary of the applicable constituent association in accordance with paragraph (c) Section 2, Article X, of the Administrative Bylaws.

- CRAWFORD, RICHARD P., Jr.
123½ Cedar Crest Dr., Auburn, Ala.
D.V.M., A. & M. College of Texas, 1957.
- DIEHL, CHESTER F.
Pipersville, Pa.
V.M.D., University of Pennsylvania, 1936.
- FORNARI, FERNANDO E. S.
19 de Mayo num. 6, Habana, Cuba.
D.V.M., University of Havana, 1942.
- GARCIA, JOSE G.
University of Miami, School of Medicine, Coral Gables, Fla.
D.V.M., University of Havana, 1938.
- GEARHART, EDWIN R.
3535 W. 4th St., Williamsport, Pa.
V.M.D., University of Pennsylvania, 1956.
- HERNANDEZ, OSVALDO N. R.
19 de Mayo num. 6, Habana, Cuba.
D.V.M., University of Havana, 1953.
- HILL, JAMES E.
12510 N. Central Hwy., Dallas, Texas.
D.V.M., A. & M. College of Texas, 1938.
- MADERAL, LUCIANO S.
Céspedes num. 21, Jaitonico, Camaguey, Cuba.
D.V.M., University of Havana, 1934.
- MANZINI, NICOLAS G.
Luz Caballero 10, Sancti-Spiritus L. V., Cuba.
D.V.M., University of Havana, 1953.
- MCCANN, PETER J.
P. O. Box 247, Shellbrook, Sask.
M.R.C.V.S., Glasgow Veterinary College, 1935.
- MOODY, ROBERT A.
3420 Delmonte St., San Mateo, Calif.
D.V.M., Ohio State University, 1938.
- NARBONA, RAUL-A. O.
Avenida 19 num. 7405, Marianao, Habana, Cuba.
D.V.M., University of Havana, 1934.
- ROBISHEAUX, JOSEPH P.
2919 South Wayside St., Houston 23, Texas.
D.V.M., A. & M. College of Texas, 1956.
- ROSS, STEWART M.
1828 E. Third St., Williamsport, Pa.
D.V.M., University of Pennsylvania, 1934.
- SCHROEDER, WILLIAM F.
Georgia Coastal Plain Experiment Station, Animal Disease Lab., Tifton, Ga.
D.V.M., Iowa State College, 1949.
- SMITH, ROBERT E.
2350 Groff Ave., Indianapolis 22, Ind.
D.V.M., Ontario Veterinary College, 1956.

Applicants—Not Members of Constituent Associations

In accordance with paragraph (e) of Section 2, Article X, of the Administrative Bylaws, the names of applicants residing outside of the jurisdictional limits of the constituent associations, and members of the Armed Forces, shall be published in the JOURNAL for two successive months.

The first notice shall give the applicant's full name, school, and year of graduation, post office address, and the names of his endorser.

First Listing

SACKMANN, HERMANN
Ostertorstr. 9a, Verden/Aller, West Germany.
D.V.M., Hannover Veterinary College, 1954.
Vouchers: H. E. Schwermann and T. Wagener.

Second Listing

SCOTT, LYLE, Jr., D.V.M., 81st Medical Detachment (VFI) APO 258, New York, N. Y.

Graduate Applicants

The following are graduates who have recently received their veterinary degree and who have applied for AVMA membership under the provision granted in the Administrative Bylaws to members in good standing of student chapters.

First Listing

A. & M. College of Texas

- BLAKEWOOD, BLAKE W., D.V.M.
Rt. 1, Old Hammond Rd., Baton Rouge, La.
Vouchers: J. K. Cooper and M. L. Hefloun.
- BURROUGHS, ALBERT L., D.V.M.
Prairiepoint, Ind.
Vouchers: W. G. Huber and J. F. Fitzgerald.
- GARZA, GILBERTO A., D.V.M.
201 E. Fifth St., Rio Grande City, Texas.
Vouchers: W. M. Romane and J. P. Davis.
- HENSON, JAMES B., D.V.M.
Tees Substation No. 3, Angleton, Texas.
Vouchers: A. I. Flowers and R. T. DuBose.
- KANE, GRADY R., D.V.M.
Box 3, Kempner, Texas.
Vouchers: J. Gandy and W. A. Roach.
- MCCRARY, JOHN H., D.V.M.
212 North Irish St., Greeneville, Tenn.
Vouchers: W. F. Sims and H. W. Hayes.
- PORTER, WALKER B., D.V.M.
815 W. State St., Groesbeck, Texas.
Vouchers: G. S. Trevino and J. N. Chastain.
- PUTTY, J. B., D.V.M.
Rt. 1, Box 777, Odessa, Texas.
Vouchers: E. R. Willmann and O. C. Collins, Jr.
- RABE, ROBERT C., D.V.M.
8300 N. Loop Rd., El Paso, Texas.
Vouchers: W. M. Romane and W. C. Banks.
- ROHEN, FRANK M., Jr., D.V.M.
818 Austin St., San Antonio 8, Texas.
Vouchers: J. N. McCanish and W. E. Hauser.
- SALBADOR, GUS W., D.V.M.
P.O. Box 323, Ronan, Mont.
Vouchers: R. C. Keyser and R. N. Read.
- VRIESENGA, JOHN R., D.V.M.
4521 San Carlos St., Dallas, Texas.
Vouchers: R. J. Beamer and G. S. Trevino.

Alabama Polytechnic Institute*

All of the following applicants, with the exception of those otherwise noted, were vouchered for by Drs. J. E. Greene and M. K. Heath.

- ADERS, BILLIE L., D.V.M.
2209 Dunstan Rd., Houston 5, Texas.
- ANTHONY, J. L., Jr., D.V.M.
Box 154, Lebanon, Tenn.
- BARBER, THOMAS L., D.V.M.
175 Fifth St., Greenport, L. I., N. Y.
- BICKHAM, ROBERT O., D.V.M.
403 E. Mag Ave., Auburn, Ala.
- BINGHAM, GENE A., D.V.M.
1640 S. Green St., Henderson, Ky.
- BLACKBURN, BILLY O., D.V.M.
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Ardmore, Tenn.
- BRYAN, MARSHALL H., D.V.M.
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Box 209, Nashville, Ill.
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8825 1st Ave., S., Birmingham, Ala.
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- HALL, HENRY W., Jr., D.V.M.
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- HAMM, PHILLIP H., D.V.M.
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55 Hamlin Dr., W. Hartford, Conn.
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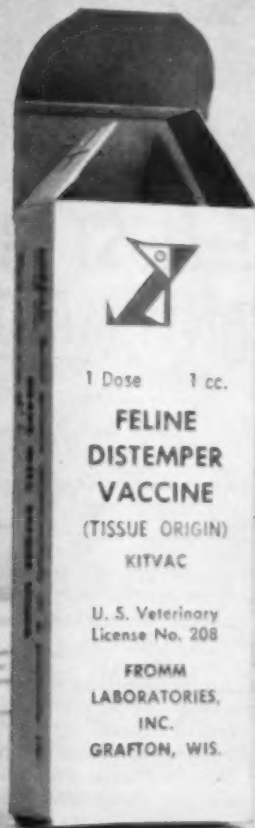
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1. Belloff, G. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956. 2. Mosier, J. E.: Vet. Med. 52:445 (Sept.) 1957.

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Make your diagnosis from the picture below—then turn the page ►



Figure 1

History.—A female German Shepherd pup, 10 weeks old, was thin and half the size of her littermates. Until she was weaned, at 4 weeks of age, she nursed and grew normally. When she was given solid food, regurgitation occurred within five minutes after each feeding, whereas milk and water were retained. She was given barium by mouth and a radiograph (lateral view) was taken.

Here Is the Diagnosis

(Continued from preceding page)

Diagnosis.—Megaesophagus.

Comment.—The problem of diagnosis is to differentiate between megaesophagus and a dilated esophagus resulting either from an obstruction, usually by a persistent aortic arch, or from spasm of the cardiac orifice (achalasia). In this radiograph, barium solution is seen throughout the entire esophagus and in the

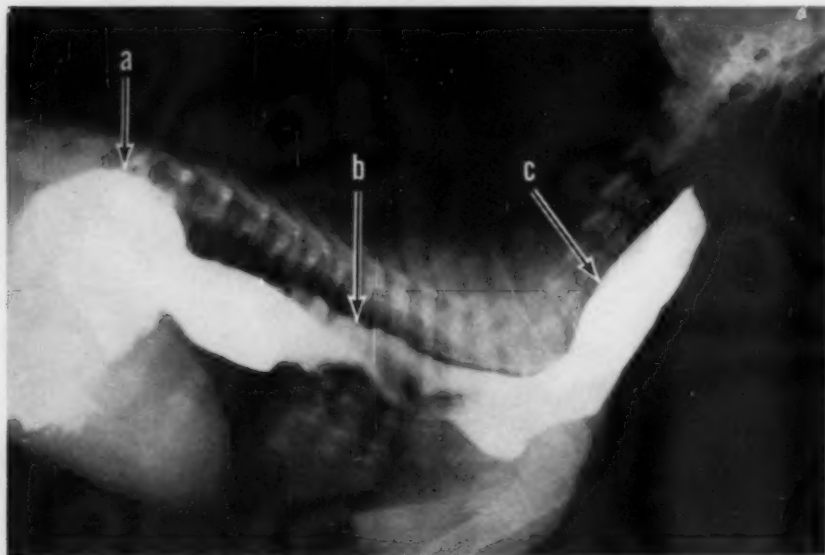


Fig. 2—Radiograph (lateral view) showing barium solution which outlines (a) the stomach, (b) the esophagus compressed at base of heart, and (c) the esophagus in the neck region.

stomach, indicating the absence of an obstruction. The narrowing of the esophagus over the base of the heart is due to the presence of the aortic arch, the pulmonary vessels, and the trachea.

This case report was submitted by members of the staff of the Yarborough Animal Clinic, Miami, Fla.

Our readers are invited to submit histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

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This film is designed to replace the 1951 release, "The Triple Threat of Brucellosis."

The film is cleared for T.V. and is recommended for showing to farm audiences.

COMING MEETINGS

New York State Veterinary Medical Society. Sixty-seventh annual meeting. Concord Hotel, Kiamasha Lake, N. Y., Sept. 4-6, 1958. Miss Joan S. Halar, 803 Varick St., Utica, N. Y., executive secretary.

Colorado Veterinary Medical Association. Annual meeting. Antlers Hotel, Colorado Springs, Sept. 5-6, 1958. G. H. Gilbert, 5500 Wadsworth Blvd., Arvada, secretary-treasurer.

New Mexico Veterinary Association. Annual meeting. La Pasada Hotel, Santa Fe, Sept. 8-9, 1958. E. R. Leslie, 920 N. Main, Carlsbad, N. M., secretary.

Washington State Veterinary Medical Association. Annual meeting. Hotel Olympian, Olympia, Sept. 8-10, 1958. F. M. Shigley, P.O. Box 1163, Olympia, Wash., program chairman.

South Dakota Veterinary Association. Annual meeting. Hotel Cateract, Sioux Falls, Sept. 16-17, 1958. G. E. Duncan, Tyndall, secretary.

Georgia Coastal Plain Experiment Station, Fourteenth annual conference for veterinarians. Animal Disease Department, Route 2, Tifton, Ga., Sept. 22-23, 1958. Maurice W. Hale, chairman.

New England Veterinary Medical Association. Annual meeting. Hotel Wentworth-By-The-Sea, Portsmouth, N. H., Sept. 28-Oct. 1, 1958. C. Lawrence Blakely, 180 Longwood Ave., Boston 15, Mass., secretary-treasurer.

Missouri, University of. Annual short course for graduate veterinarians. School of Veterinary Medicine, Columbia, Mo., Oct. 6-7, 1958. Cecil Elder, chairman.

Iowa State College. Tenth annual veterinary homecoming luncheon. Veterinary courtyard, Oct. 11, 1958, at 11:00 a.m. Robert H. Keith, 621 Pammel Court, Ames, Iowa, publicity chairman.

Florida State Veterinary Medical Association. Annual meeting. Galt Ocean Mile Hotel, Fort Lauderdale, Oct. 12-14, 1958. A. R. Chambers, 6116 Main St., Jacksonville, secretary.

District of Columbia Veterinary Medical Association. Annual all-day meeting. Walter Reed Army Medical Center, Washington, D.C., Oct. 14, 1958. W. I. Gay, 5200 Chandler St., Bethesda, Md., secretary-treasurer.

Eastern Iowa Veterinary Association, Inc. Annual meeting. Hotel Roosevelt, Cedar Rapids, Oct. 16-17, 1958. F. E. Brutsman, Trar, Iowa, secretary-treasurer.

Texas Veterinary Medical Association. Annual meeting.

Stephen F. Austin Hotel, Austin, Oct. 19-21, 1958. Paul B. Blunt, 710 Maverick Bldg., San Antonio, secretary.

Illinois, University of. Annual Illinois veterinary conference and extension short course for veterinarians. College of Veterinary Medicine, Urbana, Oct. 23-24, 1958. L. E. Boley, 311 W. William St., Champaign, Ill., chairman.

Southern Veterinary Medical Association. Annual meeting. Claridge Hotel, Memphis, Tenn., Oct. 26-30, 1958. A. A. Husman, P.O. Box 91, Raleigh, N. Car., secretary.

Mississippi Valley Veterinary Medical Association. Annual meeting. Pire Marquette Hotel, Peoria, Ill., Nov. 3-6, 1958. W. P. Hendren, Carthage, Ill., secretary-treasurer.

Arizona Veterinary Medical Association. Annual meeting. Yuma Country Club, Yuma, Dec. 7-9, 1958. Thomas Lightle, Route 1, Box 817, Yuma, Ariz., in charge of reservations.

Tennessee Veterinary Medical Association. Annual meeting. Noel Hotel, Nashville, Jan. 11-13, 1959. H. W. Hayes, 5009 Clinton Pike, Knoxville, secretary-treasurer.

Oklahoma Veterinary Medical Association. Annual meeting. Mayo Hotel, Tulsa, Jan. 25-27, 1959. M. N. Rirmensneider, 122 State Capitol Bldg., Oklahoma City, secretary.

Ohio State Veterinary Medical Association. Annual convention. Neil House Hotel, Columbus, Feb. 4-6, 1959. Harry C. Sharp, 1411 W. Third Ave., Columbus, Ohio, executive secretary.

Foreign Meetings

Sixth International Congresses on Tropical Medicine and Malaria. Lisbon, Portugal, Sept. 5-13, 1958. Professor Manuel R. Pinto, Institute of Tropical Medicine, Lisbon, secretary-general. (Membership application forms may be obtained by U.S. veterinarians by writing to the AVMA.)

Veterinary Congress of Brussels, Brussels, Belgium, Sept. 6-17, 1958. Further information available from Bureau, 35, Rue de la Brasserie, Brussels 5.

German Veterinary Association. Regular biennial meeting. Hannover, Germany, Sept. 19-21, 1958. Dr. Karl Ohly, 123 Forsthausstrasse, Frankfurt/Main, Germany, president.

Seventh International Congress on Hydatid Disease, Beirut, Lebanon, Sept. 20-22, 1958. Dr. Elias Sader, c/o Lebanese Order of Physicians, P.O.B. 640, Beirut, Lebanon, secretary-general. (Applications to present papers must be accompanied by an abstract.)

International Veterinary Congress. Sixteenth session. Madrid, Spain, May 21-27, 1959. Prof. Pedro Carda A., general secretary, Calle Villanueva 11, Madrid.

U.S. COMMITTEE: Dr. W. A. Hagan, chairman, New York State Veterinary College, Ithaca, N. Y.; Dr. J. G. Hardenbergh, secretary, 600 S. Michigan Ave., Chicago 5, Ill.

Third World Congress on Fertility and Sterility Amsterdam, Holland, June 7-13, 1959. Dr. L. I. Swaab, Sint Agnietenstraat 4, Amsterdam, Holland, honorary secretary.

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. Dr. G. W. Jones, Main St., Prattville, Ala., secretary-treasurer.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 19th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

North Alabama Veterinary Medical Association, the second Thursday of November, January, March, May, July, and September, in Decatur, Ala. Ray A. Ashwander, Decatur, Ala., secretary.

North East Alabama Veterinary Medical Association, the second Tuesday of every other month. Leonard J. Hill, P.O. Box 761, Gadsden, Ala., secretary-treasurer.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

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The pilgrims brought several dogs with them, mostly Spaniels, Terriers, a type of Shepherd, and Foxhounds, and Columbus, on his second trip to the New World, brought a type of hound trained to track enslaved natives.—*Dog World* (June, 1958): 33.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2 Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Alameda-Contra Costa Veterinary Medical Association, the fourth Wednesday of Jan., March, May, June, Aug., Oct., and Nov. Leo Goldsron, 3793 Broadway, Oakland 11, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of February, April, July, September, and December. Herb Warren, 3004 16 St., San Francisco, Calif., executive secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

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*Jones, S. V.; Belloff, G. B., and Roberts, H. D. B.: Vet. Med. 51:413 (Sept.) 1956.

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Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rocky, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643-N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. R. M. Grunfield, 2600 W. El Camino Real, San Mateo, Calif., secretary-treasurer.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert L. Chandler, P.O. Box 8, Ukiah, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossell, 1795 Moore St., San Diego, Calif., secretary.

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San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. Dr. Rolf Reese, 23815 Ventura Blvd., Calabasas, Calif., secretary.

Santa Clara Valley Veterinary Association, the fourth Tuesday of each month. Kay Beulley, N. Fourth and Gish Rd., San Jose, Calif., secretary.

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Dr. Robert N. Berkman has joined Eli Lilly & Company as a research veterinarian in the Agricultural Research Division. He will conduct investigations in pharmacology and pathology of livestock and poultry diseases, according to J. F. Downing, Ph.D., director of Lilly's agricultural research.



Dr. Robert N. Berkman

Originally a student of bacteriology, Dr. Berkman received a bachelor of science degree in 1951 and a master of science degree in 1952 from Michigan State University. Continuing his education at Michigan State, he earned a bachelor of science degree in veterinary science in 1954, a D.V.M. degree in 1956, and a Ph.D. degree in veterinary pathology in 1958.

He is a member of the AVMA; the Society of Phi Zeta, honorary veterinary fraternity; and Sigma Xi, honorary research society.



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last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinary Medical Association, the second Thursday of each month. D. E. Britten, 544 N. Ben Maddox, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

FLORIDA—Central Florida Veterinary Medical Association, the first Tuesday of each month, time and place specified monthly. Jack H. McElyer, 5925 Edgewater Drive, Orlando, Fla., secretary.

Florida West Coast Veterinary Medical Association, the second Wednesday of each month at the Lighthouse Inn, St. Petersburg. William F. Casler, 2340 30th Ave., N., St. Petersburg, secretary-treasurer.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodsons Restaurant, P. S. Roy, 4443 Atlantic Blvd., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. T. R. Geci, 108B Catherine Ave., Pensacola, Fla., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 610 Datura St., West Palm Beach. J. J. McCarthy, 500-25th Street, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Frank Mueller, Jr., 4148 E. 8th Ave., Hialeah, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. W. B. Martin, Jr., 3002 N. W. 6th St., Gainesville, Fla., secretary.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. A. E. Hixon, 131 Mary St., Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the third Thursday of each month at the Elk's Home, 726 Peachtree St., Atlanta. Donald C. Ford, Forest Park, secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Charles H. Armstrong, 1021 Davis St., Evanston, secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. Alfred G. Schiller, Veterinary Clinic, University of Illinois, Urbana, secretary-treasurer.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.



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Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P.O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Medical Association, the second Monday of each month, except January, July, August, and October in Black's Tea Room, Waterloo, Iowa. A. J. Cotten, Grundy Center, secretary.

Central Iowa Veterinary Medical Association, the third Monday of each month, except June, July, and August, at 6:30 p.m., Breeze House, Ankeny, Iowa. John Herrick, Ames, secretary.

Coon Valley Veterinary Medical Association, the second Wednesday of each month, September through May, at 7:30 p.m., Cobblestone Inn, Storm Lake, Iowa. Robert McCutcheon, Holstein, secretary.

East Central Iowa Association, the second Thursday of each month at 6:30 p.m., usually in Cedar Rapids, Iowa. Dr. J. G. Irwin, Iowa City, secretary.

Fayette County Veterinary Medical Association, the third Thursday of each month at 6:30 p.m. in West Union, Iowa. H. J. Morgan, West Union, secretary.

Lakes Veterinary Association, the first Tuesday of each month, September through May, at 6:30 p.m., at the

Gardson Hotel, Estherville, Iowa. Barry Barnes, Milford, secretary.

North Central Iowa Veterinary Medical Association, the third Thursday of April, at the Warden Hotel, Fort Dodge, Iowa. H. Engelbrecht, P. O. Box 797, Fort Dodge, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wineslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northwest Iowa Veterinary Medical Association, the second Tuesday of February, May, September, and December, at the Community Bldg., Sheldon. W. Ver Meer, Hull, secretary.

Southeastern Iowa Veterinary Association, the first Tuesday of each month at Mt. Pleasant, Iowa. Warren Kilpatrick, Mediapolis, secretary.

Southwestern Iowa Veterinary Medical Association, the first Tuesday of April and October. Hotel Chieftain, Council Bluffs, Iowa. J. P. Stream, Creston, secretary.

Upper Iowa Veterinary Medical Association, the third Tuesday of each month at 7:00 p.m., at All Vets Center, Clear Lake, Iowa. Richard Baum, Osage, secretary.

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. R. H. Folsom, P.O. Box 323, Danville, Ky., secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday of each month in Louisville or within a radius of 50 miles, except January, May, and July. G. R. Comfort, 2102 Reynolds Lane, Louisville, Ky., secretary-treasurer.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m., at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Norman Herbert, 3506 Joann Drive, Baltimore 7, Md., secretary.

MICHIGAN—Central Michigan Veterinary Medical Association, the first Wednesday of every month at 7 p.m. Frank A. Carter, P.O. Box 78, Carson City, Mich., secretary.

Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis, Mo., at 8 p.m. Edwin E. Epstein, 4877 Natural Bridge Ave., St. Louis 15, Mo., secretary.

Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Thursday of each month at the Hotel President, Kansas City, Mo. Frank A. O'Donnell, Parkville, Mo., secretary-treasurer.

NEVADA—Western Nevada Veterinary Society, the first Tuesday of each month. Paul S. Silva, 1170 Airport Road, Reno, Nev., secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

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Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Elks Club, Hackensack. Burrirt Lupton, 349 Franklin Ave., Wyckoff, secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. R. Muller, 43 Church St., Lambertville, N. J., secretary.

Southern New Jersey Veterinary Medical Association, the fourth Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. R. M. Sauer, secretary.

NEW MEXICO—Bernalillo County Veterinary Practitioners Association, third Wednesday of each month, Fez Club, Albuquerque, N.M. Jack Ambrose, 3018 N. Rio Grande Blvd., Albuquerque, secretary-treasurer.

NEW YORK—New York City, Inc., Veterinary Medical Association of the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

New York State Veterinary College, Annual conference for veterinarians. Cornell University, Ithaca. W. A. Hagan, New York State Veterinary College, Cornell University, Ithaca, N. Y., dean.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. Joseph A. Lombardo, 411 Woodlawn Ave., Greensboro, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month, time and place specified monthly. Byron H. Brow, Box 453, Goldsboro, N. Car., secretary.

Piedmont Veterinary Medical Association, the last Friday of each month. T. L. James, Box 243, Newton, N. Car., secretary.

Twin Carolinas Veterinary Medical Association, the third Friday of each month at Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. J. E. Currie, 690 N. Leak St., Southern Pines, N. Car., secretary.

Western North Carolina Veterinary Medical Association, the second Thursday of every month at 7:00 p.m. in the George Vanderbilt Hotel, Asheville, N. Car. Vilu Lind, 346 State St., Marion, N. Car., secretary.

OHIO—Cincinnati Veterinary Medical Association, the third Tuesday of every month at Shuller's Wigwam, 6210 Hamilton Ave., at North Bend Road, G. C. Lewis, Cincinnati, Ohio, secretary-treasurer.

Columbus Academy of Veterinary Medicine, every month, September through May. E. M. Simonson, Columbus, Ohio, secretary-treasurer.

Cuyahoga County Veterinary Medical Association, the first Wednesday in September, October, December, February, March, April and May, at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. F. A. Coy, Cleveland, Ohio, secretary.

Dayton Veterinary Medical Association, the third Tuesday of every month. O. W. Fallang, Dayton, secretary.

Killbuck Valley Veterinary Medical Association, the first Wednesday of alternate months beginning with February. D. J. Kern, Killbuck, Ohio, secretary-treasurer.

Mahoning County Veterinary Medical Association, the third Tuesday of each month, at 9:00 p.m., Youngstown Maennerchor Club, Youngstown, Ohio. Sam Segall, 2935 Glenwood Ave., Youngstown, secretary.

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Miami Valley Veterinary Medical Association, the first Wednesday of December, March, June, and September. J. M. Westfall, Greenville, Ohio, secretary-treasurer.

North Central Ohio Veterinary Medical Association, the last Wednesday of each month except during the summer. R. W. McClung, Tiffin, Ohio, secretary-treasurer.

Northwestern Ohio Veterinary Medical Association, the last Wednesday of March and July. C. S. Alvanson, Toledo, Ohio, secretary-treasurer.

Stark County Veterinary Medical Association, the second Tuesday of every month, at McBrides Emerald Lounge, Canton, Ohio. M. L. Willen, 4423 Tuscarawas St., Canton, Ohio, secretary.

Summit County Veterinary Medical Association, the last Tuesday of every month (except June, July, and August), at the Mayflower Hotel, Akron, Ohio. M. L. Scott, Akron, Ohio, secretary-treasurer.

Tri-County Veterinary Medical Association, the fourth Wednesday of January, May, and September. Mrs. R. Slusher, Mason, Ohio, secretary-treasurer.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Forest H. Stockton, 2716 S.W. 29th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 538 S. Madison St., Tulsa, Okla., secretary.

OREGON—Portland Veterinary Medical Association, the second Tuesday of each month, at 7:30 p.m., Ireland's

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Willamette Veterinary Medical Association, the third Tuesday of each month, except July and August, at the Marion Hotel, Salem. Marvin M. Corff, McMinnville, Ore., secretary.

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine. Raymond C. Snyder, N.E. Corner 47th St. and Hazel Ave., Philadelphia 43, Pa., secretary.

Lehigh Valley Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary.

Pennsylvania Northern Tier Veterinary Medical Association, the third Wednesday of each odd numbered month. R. L. Michel, Troy, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary Ct., Richmond 20, Va., secretary.

Northern Virginia Veterinary Conference, the second Tuesday of each month. Francis E. Mullen, 1130 S. Main St., Harrisonburg, Va., secretary-treasurer.

Northern Virginia Veterinary Society, the second Wednes-

day of every third month. Meeting place announced by letter. H. C. Newman, Box 143, Merrifield, secretary.

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. I. D. Wilson, Blackburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle, Wash. William S. Green, 9637 S. E. 36th, Mercer Island, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. O. I. Bailey, P. O. Box 906, Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Pritchard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

WISCONSIN—Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.). D. F. Ludvigson, Ridgeland, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. Dr. E. P. Pope, 409 Farley Ave., Madison, Wis., secretary.

Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. Dr. R. H. Steinkraus, 7701 N. 59th St., Milwaukee, Wis., secretary.

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In man, pain is immediate but disappears in a few minutes when a wheal, 4 to 8 mm. in diameter, appears. Within 24 hours, a pustule, 2 to 3 mm. in diameter, forms in the center of the wheal and persists for three to eight days. Persons previously stung may show an anaphylactoid reaction.

Children are treated parenterally with 1:1,000 epinephrine (0.1 to 1.0 ml.). Adults are given 1.5 ml. of injectable benadryl solution, and antihistamines are given orally to both groups.—*Pub. Health Rep.* (May, 1958): 445.



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the third Wednesday in April. William Madison, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. W. E. Lyle, P. O. Box 107, Deerfield, Wis., secretary.

Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

Wisconsin Valley Veterinary Medical Association, the second Tuesday of every other month. E. S. Scobell, Rt. 2, Wausau, Wis., secretary.

CLASSIFIED ADVERTISEMENTS

PERSONAL WANT ADS—\$4.00 for the first 25 words and 10 cents for each additional word; 35 cents for use of box number.

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COMMERCIAL WANT ADS—\$5.00 for the first 25 words, 25 cents for each additional word; \$1.00 for use of box number. (See paragraph above for total word count.)

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DEADLINES

1st of month issue — 8th of month preceding date of issue.

15th of month issue — 22nd of month preceding date of issue.

Names of classified advertisers using key letters can not be supplied. Address your reply to the box number, c/o JOURNAL of the AVMA, 600 S. Michigan Ave., Chicago 5, Ill., and it will be sent to the advertiser.

Wanted—Veterinarians

Wanted—career state civil service field veterinarians, \$6,444-\$8,424, vacation, sick leave, retirement benefits. Ages 21-50, graduate accredited college; U.S. citizenship required. Contact Animal Health Division, State Capitol, Madison, Wis.

County livestock inspector—\$677 to \$842 per month. Must possess a valid license to practice as a veterinarian in California and have four years of experience practicing veterinary medicine. For applications and information write Orange County Personnel Office, 801-C North Broadway, Santa Ana, Calif.

Wanted—experienced veterinarian to take charge of mixed practice, predominantly small animal. State age, description, qualifications, expected salary, and three references. Apartment available. Address "Box P 1," c/o JOURNAL of the AVMA.

Veterinarian wanted—to assist in mixed practice (mainly range cattle) in upper range state. Please state qualifications and salary expected. Address "Box P 3," c/o JOURNAL of the AVMA.

Associate veterinarian wanted in mixed practice, 60 per cent small animal, middlewestern metropolitan area. Salary and commission; good prospects. Address "Box P 4," c/o JOURNAL of the AVMA.

Veterinarian at once to assist in mixed practice. Modern small animal hospital, sale barn, and large animal work. Auto required. Contact Dr. Elmer F. Goetz, 709 South 18th, Mattoon, Ill.

Veterinarian—ethical nutritional supplement house located Middle West desires veterinarian to serve as consultant, distributor contact man, and salesman. Car furnished. Address "Box P 12," c/o JOURNAL of the AVMA.

Wanted—capable veterinarian of accredited school to assist in a small animal hospital in Pennsylvania. State age and qualifications. Furnished apartment available. Address "Box P 16," c/o JOURNAL of the AVMA.

Experienced veterinarian capable of taking complete charge of a busy small animal hospital. Must have Connecticut license. Excellent opportunity leading to partnership or purchase. Write complete history and experience. Address "Box P 19," c/o JOURNAL of the AVMA.

Wanted—Positions

Veterinarian desires new appointment. Background in biological research, development and production; diagnostic laboratory, research, and teaching experience. Good reason for change. Address "Box P 7," c/o JOURNAL of the AVMA.

Relief veterinarian, experienced and competent, will operate your practice while you vacation. Available New York City and suburbs. Licensed in New York and Connecticut. Address "Box P 9," c/o JOURNAL of the AVMA.

Texas graduate ('56) desires position large animal or mixed practice. Married, age 25, energetic, capable, completes military obligation October. Prefer southwestern or central U.S. Licensed Texas, New Mexico. Address "Box P 11," c/o JOURNAL of the AVMA.

Wanted—Practices

Veterinarian—experienced small animal practitioner wishes to purchase a well-established practice and physical plant. Capital available for right deal. Age 31, married. Address "Box P 14," c/o JOURNAL of the AVMA.

Experienced veterinarian (OSU), married, children, desires to lease or purchase small animal hospital. Excellent references; any area considered. Address "Box P 15," c/o JOURNAL of the AVMA.

Southern California—want established small animal hospital for lease with option to buy. Five years' local experience. Address "Box P 17," c/o JOURNAL of the AVMA.

For Sale or Lease—Practices

Prosperous, well-established small animal hospital, New York State. Includes valuable real estate, 4-bedroom home. Sacrificing for quick sale. Address "Box M 33," c/o JOURNAL of the AVMA.

For rent—established Connecticut mixed practice for rent on a percentage basis. Eventual ownership small animal hospital. Address "Box P 8," c/o JOURNAL of the AVMA.

For sale—well-established, active mixed practice in New York State, high class suburb of New York City. Gross \$30,000, half small animal. State, county, and township work available. Practice could be enlarged in either large or small animal as desired. Completely-equipped small animal hospital, exclusive clientele. Colonial-type residence, modernized, on 2½ acres of land beautifully landscaped. Owner has practiced in this location for 25 years and wants to retire from practice. Present veterinary assistant willing to work with new owner. Priced reasonably. Address "Box M 31," c/o JOURNAL of the AVMA.

For sale—in intermountain area, well-established mixed practice. Two acres, ranch-type brick home, attached clinic, drugs, and equipment, including 2-way radio. Address "Box P 2," c/o JOURNAL of the AVMA.

For sale—long-established fashionable small animal practice. Beautiful hospital, home, and grounds; located east central New England. Owner desires to retire. Address "Box P 5," c/o JOURNAL of the AVMA.

Fully-equipped hospital and apartment in heavily populated section of Chicago. \$25,000; \$5,000 down. Will consider lease; \$350 including apartment. Give personal and credit references in first letter. Address "Box P 6," c/o JOURNAL of the AVMA.

Established large animal practice in southern Michigan. Owner going into partnership. Low investment—drugs and house equity. A wonderful opportunity for recent graduate who desires good practice with a low investment. Address "Box P 10," c/o JOURNAL of the AVMA.

Dairy cattle practice in east central Wisconsin includes 7-room modern home with office and mobile radio equipment. Grossed \$25,000 last year. Needs \$5,000 cash to handle. Address "Box P 13," c/o JOURNAL of the AVMA.

For sale—large animal practice in Illinois grossing approximately \$30,000. Drugs and equipment; no real estate. Address "Box P 18," c/o JOURNAL of the AVMA.

Too Late to Classify

Wanted—veterinarian for Chicago small animal hospital, to assist two other veterinarians. Apartment available. Include college and year of graduation, experience, references, salary expected in application. Address "Box P 20," c/o JOURNAL of the AVMA.

To insure prompt delivery, replies should be carefully addressed: Complete box number as given in the ad, AVMA, 600 S. Michigan Blvd., Chicago 5, Ill.



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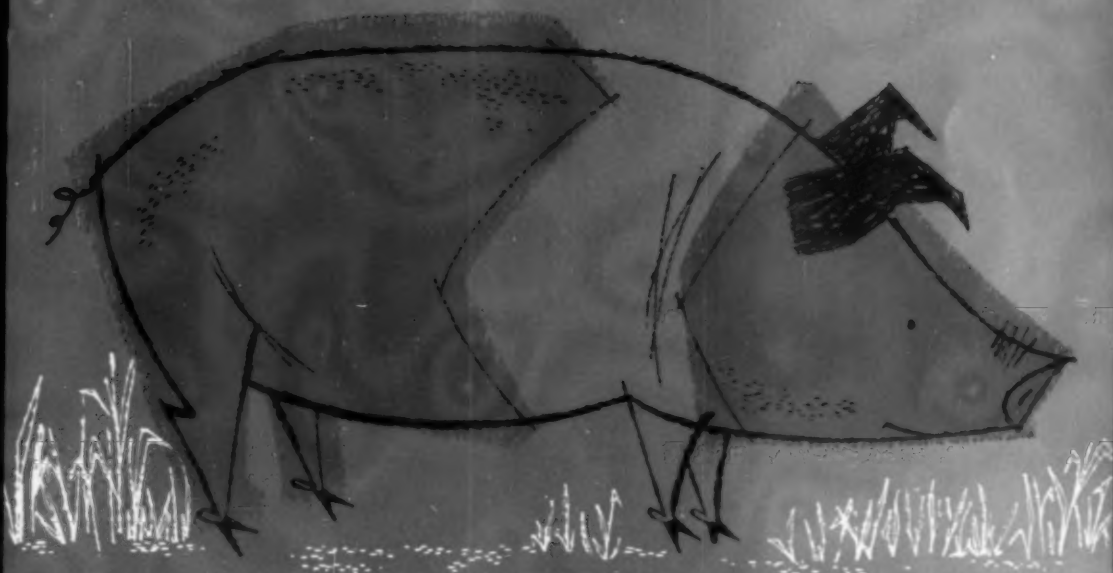
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